APR 12 1927

Volume IX APRIL, 1927 Number 4

THE JOURNAL OF INDUSTRIAL HYGIENE

AND ABSTRACT OF THE LITERATURE

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The Silicosis of the South African Gold Mines, and the Changes Produced in It by Legislative and Administrative Efforts

Experimental Electric Shock

Published Monthly at Mount Royal and Guilford Aves., Baltimore, Md.
by HARVARD SCHOOL OF PUBLIC HEALTH

Entered as second-class matter, at the Post Office at Baltimore, Maryland, under Act of March 3, 1879. Published Monthly.
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PUBLISHED MONTHLY

VOLUME IX APRIL, 1927 NUMBER 4

THE SILICOSIS OF THE SOUTH AFRICAN GOLD MINES, AND THE CHANGES PRODUCED IN IT BY LEGISLATIVE AND ADMINISTRATIVE EFFORTS*

W. WATKINS-PITCHFORD, M.D. (LOND.), F.R.C.S. (ENG.)

Director of the South African Institute for Medical Research; Chairman of the Miners' Phthisis Medical Bureau, Johannesburg; Hon. Professor of Pathology and Bacteriology, University of the Witwatersrand

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* Written Feb. 15, 1926. Received for publication Aug. 31, 1926.

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Local Circumstances

SILICOSIS has been produced on the Rand in the course of mining through quartzite (containing 90 to 96 per cent. of free silica) in order to reach, remove, and crush the bands, or reefs, of gold containing conglomerate; this conglomerate contains about 86 per cent. of free silica, and consists of quartz pebbles embedded in a silicified matrix.

The same quartzite forms the common surface rock over large areas of the Transvaal, and silicosis is occasionally developed as the result of repeated exposures to the dust storms of the dry season. Thus, among 34,000 candidates for the "Initial Examination" of the Medical Bureau, no less than seven well-marked cases of silicosis have been found in men—farmers, policemen, and transport riders—who have never engaged in mining or stonecutting. It will therefore be seen that silicosis, like many other occupational diseases, is not strictly confined to particular industries.

It is probable that no industrial disease in any country has received so much legislative attention, from the compensatory aspect, in such a short space of time. The Union of South Africa was constituted in 1910; and between the years 1911 and 1925 its Parliament has passed no fewer than nine Acts, principal, amending, or consolidating, and dealing mainly with the compensation to be paid to workers, or the dependents of deceased workers, who have contracted this disease and/or pulmonary tuberculosis. The justification for this legislative activity will be apparent when it is remembered that the production of cases of silicosis has been associated almost *pari passu* with the progress of the gold mining industry, from a few years after the sinking of the first shaft until the present time. From its beginning in 1886, the industry in the Transvaal has undergone an extraordinarily rapid development, so that by the year 1923 it was supplying more than 50 per cent. of the total output of gold for the world. During the thirteen years 1911 to 1923 the mines of the Rand produced an annual average of more than thirty-five million pounds' worth of gold. That the creation of such colossal amounts of wealth was associated with the production of a very serious disease of the lungs in a considerable proportion of the workers was a fact which the legislature of a civilized country could not possibly ignore. A Commission in 1912 found that about 26 per cent. of the European miners were affected with silicosis, while during the period 1916 to 1924 there has been an annual production, on the average, of about 300 cases of this disease among the European miners, and 380 among the native laborers. In the period last mentioned the number of working miners has averaged about 15,000, and the native laborers about 175,000.

The all-important matter of suppressing, or limiting, the creation of dust in the mines, and protecting the workers from inhaling the dust, has been solely provided for by certain

of the terms of a single Act—the Mines and Works Act of 1911; the pertinent Regulations under this Act have, however, been amended and enlarged from time to time.

In 1916 a Medical Bureau was appointed by the government to examine all candidate miners as to their physical fitness (the "Initial Examination"); to examine at intervals of six months all working miners for the presence of pulmonary tuberculosis or of silicosis (the "Periodical Examination"); to examine all ex-miners claiming to be silicotic (the "Benefits Examination"); to supervise the examinations of the native laborers; and to certify as to the presence of silicosis, and whether it was the cause, or a contributory cause, of death in any mine worker.

The present occasion is not appropriate for a detailed review of all the researches which have been conducted into the causation and pathology of the disease; it will suffice to embody the results of the more important of these researches (with special acknowledgments to Dr. A. Mavrogordato and Dr. L. G. Irvine) in a short account of the disease as we have encountered it in the Transvaal. One feels a certain amount of assurance in undertaking this task, and some confidence that it will prove of service in other countries, because of the unusually large number of cases met with in Johannesburg, and because of the unique facilities which have been afforded, for many years past, for systematic observation and study of the disease in all its aspects. The extent of these facilities may be gauged from the fact that the Medical Bureau, since 1916, has conducted, on the average, 34,890 examinations annually; has prepared and examined, in each year, 24,568 radiographs of the chest; and has annually carried out 342 postmortem examinations of the lungs of deceased mine workers.

Name of the Disease

The name "silicosis" is more acceptable to the medical scientist than "miners' phthisis," since it indicates the primary cause of the disease and not merely one of its symptoms. "Phthisis," *i.e.*, wasting, is not necessarily present, and, moreover, it is a symptom presented by many other ailments of very varied causation. The South African Miners' Phthisis Act of 1912, and all its principal successors, have happily declared that "miners' phthisis means silicosis of the lungs;" and the term silicosis is now lawfully applied to designate any and all of the varieties of the disease, whether an obviously tuberculous process is present or not. The term miners' phthisis, if it has any precise meaning, refers only to silicosis when associated with an active tuberculous process; its retention in the title of the South African Act, and in the title of the Bureau which administers its medical provisions, is indeed merely a historical relic.

Causation

The silicosis of the South African gold miner is etiologically identical in all its essentials with tin miners' phthisis, stone cutters' phthisis, potters' rot, and grinders' disease; these latter diseases appear to be examples of that form of silicosis which is known to us as "silico-tuberculosis," and which will be subsequently described.

The primary factor in the causation of all these diseases is the entry into the lymph channels of the lung, *via* the air vesicles, of excessive quantities of particles of crystalloid silica—usually in the form of quartz, sandstone, or flint. These particles eventually tend to become incarcerated in new fibrous tissue in the lung, but this process of incarceration is usually interfered with by a secondary factor which is of paramount importance in the disease—*viz.*, the activities of the *Bacillus tuberculosis*. This microbe may be already present in the lung, it may enter it almost simultaneously with the silica particles, or, as is more commonly the case, it obtains lodgment subsequently.

The mere presence of a few particles of silica dust in the lung, although probably predisposing to pulmonary tuberculosis, is not sufficient to give rise to silicosis, at least in its common and generalized form; but no definite statement can be made as to the degree of abundance of particles which is prerequisite to the development of the familiar disease. The lungs of people in the Transvaal who have not followed any dusty trade, and who were certainly not silicotic, have been found to contain as many as 100,000 million particles of silica dust. On the other hand, the number of such particles in the lungs of a silicotic miner must certainly be reckoned in millions of millions. In at least the very great majority of cases, the final and determining cause of the appearance of the disease in the Rand gold miner, even in its "simple" form, has been a tuberculous infection, rather than a surpassing of the capacity of the lung harmlessly to incarcerate quartz particles. One suspects that the same observation will be confirmed in all the silicosis producing industries.

The size of the silica particles (mostly from 0.5 micron to 5 microns) which gain access to the lung tissues is limited by the capacity of the phagocytic endothelial cells, lining portions of the air vesicles, to engulf them and convey them from the vesicles into the lymph spaces and lymph channels of the lung. It is probable that particles below about 0.25 micron, or above about 10 microns, are practically innocuous, since they cannot be phagocytosed.

Foreign particles of any sort, provided they are of a size falling within the limits just stated, will be conveyed into the lymph spaces of the lung substance if they have been inhaled in sufficient numbers. With the exceptions of silica and pathogenic microbes, however, almost all other foreign particles are practically harmless; in proportion to their insolubility, they tend to loiter and accumulate, for varying lengths of time, in the lymphatic spaces, channels, and nodes. They thus give rise to such nonpermanent and relatively harmless conditions—one can hardly call them diseases—as anthracosis, aluminosis, siderosis, etc.

Silica particles which have been carried into the lymphatic system of the lung are specifically harmful in two ways: (1) They eventually become encapsuled in small areas of fibrous tissue, and when these areas are very numerous and widely distributed a certain amount of mechanical interference with the functions of the lung results (simple silicosis); (2) their presence greatly facilitates the multiplication of the tubercle bacillus,

if this also gains entry to the lymph spaces (tuberculosis with silicosis). Both these effects of silica are probably chemical in nature; the first is slowly developed during the very gradual solution of the particles, while the second is evidenced without delay.

If the particles are of silicate—*i.e.*, silica combined with metallic oxides—such as glass, brick, or cement, the specific effects of silica are not produced.

It is only a small proportion of those who have been exposed to the specific dust for similar periods who develop silicosis. Thus, in the year 1924, of 2,302 European miners who had worked underground on the Rand for ten years or longer, only 176 (7.6 per cent.) developed silicosis in any form during the year; the remainder (92.4 per cent.) were apparently quite free from the disease. That this was not merely a result of inability to diagnose the disease during life is shown by the fact that, among the postmortem examinations, during the same year, of the lungs of miners who had also worked underground for more than ten years, no trace of silicotic fibrosis could be found in six instances.

That a very large proportion of the men employed for the same periods in similar classes of work have failed to develop the disease may be accounted for by one or both of the following suppositions: (1) that they have escaped the tuberculous infection which, in their less fortunate fellows, is the final excitant of the disease; and (2) that, for one reason or another, the amounts of dust actually inhaled have been relatively smaller; for experimental observations on animals show that, if the exposures to silica dust are sufficiently intense, no individual will escape the subsequent development of a nodular pulmonary fibrosis. When the quantity of dust inhaled by the miners has been relatively small, it may be surmised that the processes whereby the particles are removed from the lungs have been sufficiently active, in the majority of the men, to prevent that degree of accumulation of particles which is prerequisite to the development of the ordinary form of silicosis.

Latent Silicosis

Despite the nondevelopment of silicosis in the great majority of those who have been exposed to the dust for a long period, it appears that the *liability* to such development may nevertheless be present in many of the individuals. It was found, for example, that several of the long-service miners who took part in the Great War, and who were non-silicotic when they left their work, had developed the condition when they returned. This state of liability doubtless depends upon the continued retention in the lung tissues of a sufficient number of silica particles; it ceases when a certain proportion of them have escaped in the expectoration, have been removed by other channels, or have become finally dissolved. The agency which converts the liability into a manifestation of the disease, in any particular case, is usually a tuberculous infection. The state of the individual thus liable to develop the disease without inhaling any more silica dust is conveniently referred to as latent silicosis.

It will thus be seen that one has come to the conclusion that, in practically all instances, there are two fac-

tors which are prerequisite to the development of silicosis in its usual form: (1) the condition of latent silicosis, and (2) the access of a tuberculous infection.

The condition of latent silicosis presents no specific physical signs, or symptoms, and cannot therefore be diagnosed with certainty during life. It is probably associated, radiographically, with some increase of the shadows—both dendritic and reticular—normally cast by the connective tissue of the lungs, and, from the pathologist's point of view, with excess of pigmentation on the cut surface of the divided lung.

Clinical Forms of Silicosis

Silicosis is recognized in the mine workers of the Rand in one or other of two forms—either as simple silicosis or as tuberculosis with silicosis—and these two forms are separately provided for in the Miners' Phthisis Act. The fundamental distinction between the two forms is that simple silicosis is not *obviously* associated with the evidences of tuberculous infection. We are not now concerned with ordinary pulmonary tuberculosis, although it occurs among the mine workers of the Rand (especially the native laborers) and is also a legally compensable condition; mention is now made of it because, when it is discovered without any signs of coexistent silicotic changes, it is referred to, for purposes of discrimination, as "simple tuberculosis."

Although both forms of silicosis are characterized by the presence in the lungs of nodular lesions, they can be readily distinguished from one another, especially as a result of the differences between their radiographic appearances.

Simple Silicosis

Simple silicosis is a condition which, in a worker who remains free from overt tuberculous changes, is nonprogressive and produces but little effect on health. The typical condition is associated with the presence of *numerous*, small, inert nodules of dense fibrous tissue, *scattered symmetrically and uniformly* throughout the parenchyma of both lungs. When these nodules are so abundant as to be closely aggregated, a certain amount of disability may result; the leading features of this disability are shortness of breath on exertion and some tendency to obesity. When the nodules are few and widely separated, their presence can hardly be deemed to constitute a disease, and their existence indeed often goes undetected during life. The pathologist who discovers these scanty lesions will report the presence of "a few simply silicotic nodules," but he will not certify that the individual was the subject of silicosis. The convention of the Medical Bureau as to what degree of prevalence of these nodules justifies the opinion that the disease simple silicosis is present, is recorded in a subsequent section.[1] In this connection it may be remarked that patient microscopic investigation will usually disclose, in the lungs of *any* adult who has resided in the Transvaal for several years, the presence of a few small areas of fibrosis surrounding particles of silica dust and pigment;

[1] See under "Detection of Silicosis after Death," p. 124.

this widespread occurrence of miniature silicotic nodules would not, of course, justify the assertion that most of the adults in the Transvaal are affected with the disease known as silicosis.

Tuberculosis with Silicosis

Tuberculosis with silicosis (or "infective" silicosis, as it has been felicitously termed) is a progressive and very serious disorder. It is associated with the presence in the lungs of nodules of somewhat larger size, unsymmetrically distributed in the two lungs, and composed of young connective tissue elements among which the *Bacillus tuberculosis* is harbored; adjacent nodules are frequently aggregated into masses. The condition gives rise to advancing disability and cachexia.

When silicosis first appears in the European miner it is usually in the form of simple silicosis, while in the native laborer it usually originates as tuberculosis with silicosis; thus, of all new cases among the miners during the last seven years, 92 per cent. have started as simple silicosis, while of all cases among the laborers, in the same period, 78 per cent. have been in the form of tuberculosis with silicosis. The principal influence producing this racial discrimination is the much greater susceptibility of the Negro to the effects of tuberculous infection. The general question of racial liability to silicosis will be again referred to later.

True simple silicosis undoubtedly exists as a nosologic entity, for it can readily be produced in animals; while necropsies in cases of violent death have showed that it occasionally occurred among our miners in the past, when exposures to dust were often very intense. In my opinion, however, the pathology of almost all the cases of what we term simple silicosis now appearing on the Rand is fundamentally the same as that of tuberculosis with silicosis; the development of what appears to be a condition of simple silicosis, in these days, is probably a pathologically unique result of a tuberculous infection in a latently silicotic individual.

Although the pathology of the two forms of silicosis, as encountered during late years, may be fundamentally the same, the subsequent history of the two classes of cases clearly shows that their separation is clinically justifiable, and that, at least in the matter of prognosis, the two conditions are quite distinct. The prognosis in the different forms of the disease will be dealt with subsequently.

Tuberculo-Silicosis and Silico-Tuberculosis.—From the clinical standpoint, and especially in the matter of radiographic appearances, cases of tuberculosis with silicosis fall into two classes: (1) those in which an apparently simple silicosis has first declared itself, and in which, at some later date, signs of overt tuberculous changes are developed, unsymmetrically, among the silicotic lesions—as far as the European miners are concerned, this is markedly the larger class, and the condition is suitably known as "tuberculo-silicosis;" (2) those in which the signs first appearing are those of ordinary pulmonary tuberculosis, and in which the signs of a more or less localized silicosis are developed shortly afterward (or are found postmortem) in close associa-

tion with the tuberculous lesions. This condition, which is relatively more prevalent among the native laborers, is known as "silico-tuberculosis." The division of cases of tuberculosis with silicosis into these two classes has the advantage of indicating, from the clinical point of view, the factor which is primary, and dominant throughout, in each class. It should be clearly understood, however, that the discrimination between tuberculo-silicosis and silico-tuberculosis is made solely upon clinical grounds, and that it does not imply any essential difference in the fundamental pathologic processes involved.

The frequency with which the condition of simple silicosis passes into that of tuberculo-silicosis will be discussed in the next section.

The condition of silico-tuberculosis is a very important one from the legal point of view, and therefore calls for special consideration. Pathologic investigation shows that, in the majority of European cases of the condition, the tuberculous trouble has extended from one or other of a few old silicotic lesions, in the interior of which the infection has apparently been locked up for a long time. This observation suggests that silico-tuberculosis in these cases is, from the pathologic aspect, essentially the same process as tuberculo-silicosis, and that the clinical differences between the two conditions are merely consequent upon the relative abundance or scantiness of the preexisting silicotic lesions. This leads to the important conclusion that, although simple silicosis cannot be said to exist unless the lesions are multiple and scattered throughout both lungs, the condition of silico-tuberculosis must be admitted to be present even when the tuberculous process was initiated by a solitary infected silicotic lesion.

In native cases of silico-tuberculosis, however, a previously infected silicotic focus is rarely found; the tuberculous infection has been recently acquired, and has resulted in a more or less localized development of young silicotic lesions of a peculiar character.[2]

In both European and native examples of this condition, the quantities of dust previously incarcerated in the lungs have been insufficient to provide for the appearance of the numerous and extensively distributed lesions which usually constitute simple silicosis and tuberculo-silicosis.

It will perhaps be found that a majority of cases of the "phthisis" which characteristically affects certain trades, other than mining, and in which there is some exposure to silica dust, are, in fact, cases of silico-tuberculosis. This point can be cleared up only by extensive and systematic resort to post-mortem examinations; the undertaking of such a research is justified by the conclusion just referred to, and for the sake of emphasis this conclusion may be restated: Unlike simple silicosis and tuberculo-silicosis, silico-tuberculosis may appear in a lung in which but small amounts of silica dust have been incarcerated, or in which the original simply silicotic lesions were relatively very few in number, or even solitary.

Liability of Simple Silicosis to Become Overtly Tuberculous

The condition of simple silicosis is unfortunately liable to pass into that of

[2] See under "Changes Which Characterize Tuberculosis with Silicosis," p. 120.

tuberculo-silicosis, and this even after several years. In view of the gravity of this change, any observations which help us to assess the probability of its occurrence are of special value. On reviewing the subsequent histories of each annual batch of new cases of simple silicosis, it is found that the liability to the development of a tuberculous change has not been the same in each year's quota, but that it has tended, slightly but quite definitely, to decrease in the quota for each succeeding year. Without discussing the numerous and important questions which this phenomenon evokes, it may be stated that one has come to the following significant conclusion: that the liability of simple silicosis to become overtly tuberculous is mainly dependent upon a continued activity in the lung of those factors which were responsible for producing the original condition; in other words, that the major part of the liability to the complication is inherent in the case from the outset, and that the influence of subsequent environment is of secondary importance.

In ascertaining, from our records, what the liability of the simply silicotic miner to become tuberculous has been in the past, account has of course been taken of all instances in which the change has been detected during life; in addition to this, however, it has been assumed that, in those persons suffering from simple silicosis who have died of their disease without having been again examined, death has been the result of this change. This is an assumption which is warranted in practically every instance. Relying on this assumption, and also bearing in mind that, as already stated, the liability to tuberculous change has appeared less marked in each succeeding annual batch of cases, one finds that the combined results of seven years' observations show that, by the end of the seventh year, a little more than half of the cases of simple silicosis have either developed overt tuberculous changes or have died as the result of those changes (see Table 1).

TABLE 1.—LIABILITY OF SIMPLE SILICOSIS, IN EUROPEAN MINERS, TO PASS INTO TUBERCULO-SILICOSIS

(Rates per thousand of original cases of simple silicosis)

YEAR FOLLOWING THAT IN WHICH SIMPLE SILICOSIS FIRST APPEARED	LIVING, BUT NOW TUBERCULO-SILICOTIC	DEAD OF SILICOSIS	TOTAL RATE FOR OCCURRENCE OF THE CHANGE
1st	59	24	83
2nd	83	55	138
3rd	120	92	212
4th	141	139	280
5th	179	191	370
6th	217	262	479
7th	246	294	540

Apparent Immunity of the Wife

No instance is known to the Medical Bureau of a wife having developed an overtly tuberculous affection as the result of receiving infection from her silicotic husband, even though she may have successively married and nursed several victims of the disease. This observation is, of course, as old as Georgius Agricola (1556), and has been made in England, in modern times, in connection with the grinding industry

and the workers in hard sandstone quarries. The financial encouragement to matrimonial speculations of this sort was abolished by the Miners' Phthisis Act of 1919, which provided that the pension of the widow of a silicotic miner shall cease if she remarries.

This immunologic phenomenon, combined with certain others, has most mischievously obscured the communicable nature of this dreadful infection. Whatever may be the real nature of the apparent immunity of the wife, it does not seem to be due to any lack of virulence in the *Bacillus tuberculosis* which destroys the life of the husband; the sputum of the overtly tuberculous silicotic has been found to give rise to fatal tuberculosis, in susceptible animals, with just the same certainty and rapidity as that from a case of ordinary pulmonary tuberculosis.

Changes Which Characterize Simple Silicosis

When a particle of silica has been engulfed by an endothelial cell of an air vesicle, and transported into the perivascular lymph spaces, the first characteristic change is the coating of the particle with fine granules of a loosely adherent black pigment. This production of pigment is an essential phenomenon in the silicosis of the Rand, and is perhaps constant in all human silicosis. Unless at least some excess of pigmentation is found in the substance of the lungs of a deceased Rand miner, it may be safely concluded that he was not the subject of the disease in any form. In silicotic lesions in the pleura, however, pigmentation is usually slight, or absent; while the lungs of animals (rats and guinea-pigs) which have developed experimental silicosis may be but scantily, if at all, pigmented.

The next event is the death of the "dust cell" which has conveyed the silica particle, with the result that the particle and the pigment are liberated. The particle is again engulfed, probably on several occasions, either by wandering endothelial cells or by fixed connective tissue corpuscles, and the process of pigment production and the death of the cell is repeated in each instance.

The occurrence of these events throughout both lungs results in the deposition of visible black pigment in the areolar coats of the smaller blood vessels and air tubes, thus giving rise to the appearance of a pigmentation of dendritic distribution.

The particles and pigment now tend to accumulate at certain points in the lymph channels, and as soon as a sufficient number of particles have thus collected, a visible island of pigmentation is produced; so that, in the individual who has inhaled much silica dust, the parenchyma of both lungs has now become uniformly stippled with these islands of pigmentary deposit.

Ultimately, some or many of these collections of particles and pigment become surrounded and infiltrated with new fibrous tissue, thus giving rise to immature "simply silicotic nodules." In the pleural lymphatics these deposits of fibrous tissue are not usually associated with visible pigment, but appear as small whitish areas of a circular or irregular outline. The particles which have been conveyed to the bronchopulmonary lymph nodes

produce in those organs a dense and pigmented fibrosis, without, however, any increase in size.

The number and size of the pigmentary deposits, and of the pigmented nodules, in the lung depend upon the amount of fine silica dust which has gained entrance. Their distribution is symmetrical and uniform throughout both lungs, for there is usually no reason, in the absence of tuberculosis, why the invading dust should not be uniformly distributed.

It is a somewhat remarkable fact that, although silica particles may have been entering the lung substance during several years—beginning to produce pigmentary deposits from the outset—the development of the silicotic nodules may be much delayed. These latter are rapidly produced, either within a short time or simultaneously, in their maximum numbers throughout the lungs. Thereafter, a coalescence of small adjacent nodules takes place, the nodules thus becoming fewer in number but larger. In its final form, each nodule is sharply defined from the surrounding tissues, and possesses a spherical or oval contour. It is usually from 3 mm. to 4 mm. in diameter, and very rarely exceeds 6 mm. So completely do these simply silicotic nodules become encapsuled that the pathologist can often shell them out of the lung like gray or black pearls. After the lapse of many years, and in the continued absence of tuberculous infection, these nodules tend to disappear—the fibrous tissue becomes absorbed, while such of the minute silica particles as have escaped complete solution eventually find their exit from the body.

Changes Which Characterize Tuberculosis with Silicosis

From the very nature of things, one would expect to find certain pathologic features common to both simple silicosis and that form of pulmonary tuberculosis which is originated by inhaling the specific bacilli. The invading particles in the two conditions are of the same order of magnitude; they are both engulfed by endothelial cells lining the air vesicles, and carried into the same lymph spaces and channels of the lung; and in both instances the organism attempts to incarcerate the foreign particles by embedding them in new tissue. The main points of difference between ordinary bronchopneumonic pulmonary tuberculosis and simple silicosis are that in the former (1) the foreign particles have been capable of enormously multiplying themselves after gaining entrance to the pulmonary lymphatics; (2) the new tissue which is formed to shut off the particles consists mainly of endothelial cells which are very prone to death in the presence of the bacillary toxins; and (3) the areas of new tissue produced are much more extensive and are not sharply defined from their surroundings—they merge gradually into them through a zone of lymphocytes.

It is not necessary to detail the changes which characterize the three forms (bronchopneumonic, pneumonic, and miliary) of ordinary pulmonary tuberculosis, for pathologists throughout the world are but too familiar with them. It is, however, of some importance to note that, in a man who is the subject either of latent silicosis or of a well-developed simple silicosis,

a superadded tuberculous infection of the lungs does *not* give rise to the familiar lesions of ordinary tuberculosis, and particularly that the characteristic tubercles are conspicuously absent. The only exception to this general statement is that miliary tuberculosis, in its familiar form, may very occasionally affect the simply silicotic lung. A tuberculous infection in a lung which is latently or simply silicotic usually produces changes which are distinctive, in fact pathognomonic, of the condition known as tuberculosis with silicosis.

In this condition, as in simple silicosis, there is always an excess of pigmentation, but the islands of pigmentation are no longer distributed uniformly throughout the two lungs; they tend to be more numerous in the immediate neighborhood of the specific lesions of tuberculosis with silicosis. These lesions, unlike the nodules of simple silicosis, are distributed with marked irregularity and asymmetry, and, in the condition of silico-tuberculosis, may be very few in number, or even solitary.

The specific lesions of tuberculosis with silicosis result from an infiltration of the interalveolar, perilobular, perivascular, and peribronchiolar lymph spaces and channels with young and exuberant connective tissue, composed mainly of spindle-shaped cells. To the unaided eye this new tissue appears succulent and steel gray in color, and usually stands out in marked contrast with the pigmented and congested background of the cut surface of the lung. It presents itself in the form of small circles, surrounding the divided vessels, and as larger and more or less circular outlines, or groups of outlines, indicating lobules and groups of lobules. The areas enclosed within these lobular outlines are more darkly pigmented and are irregularly divided by partitions of the gray tissue. To the patterns thus produced upon the cut surface of the lung affected with tuberculosis with silicosis one has applied the name "fibrotic figures."

When isolated lobules are affected in this manner they constitute what are known as "tuberculo-silicotic," or "silico-tuberculotic," nodules—according as silicotic lesions prevail throughout both lungs, or are confined to a few localities. These nodules vary in diameter from 3 mm. to 8 mm.; but, as it is more usual for many adjacent lobules to be simultaneously affected, masses of considerably larger dimensions are commonly produced. To such masses the term "fibrotic consolidations" has been applied.

Pleurisy in a silicotic individual is usually indicative of the presence of a tuberculous infection, and this may result, more especially in the Negro, in enormous thickening of the pleurae owing to infiltration of their lymphatics with the new gray tissue.

In individuals in whom the resistance to tuberculous infection is very low, such again as South African Negroes, the infiltration of parts of the silicotic lung with this new tissue is so rapid that it may render large areas dense, airless, and friable, with a cut surface which has somewhat the appearance of gray granite, and which shows little or no trace of lobular outlines.

This young, gray, fibrous tissue which is so characteristic of tuberculosis with silicosis is very prone to

necrosis, and in this way vomicae are produced, especially in the central parts of masses of fibrotic consolidation. The bronchopulmonary lymph nodes, if already completely fibrosed by simple silicosis, may show no other change; if, however, the fibrosis has not been complete, they may be found to be enlarged and presenting areas of caseation.

In the lungs of long-surviving cases —those in whom the amount of dust in the lungs was not excessive, and in whom the superadded tuberculous infection has been more or less overcome—the former site of an area of fibrotic consolidation will appear as a dark gray or black, airless, tough, fibrous mass.

It is of some interest to note that, in fatal cases of tuberculosis with silicosis, tuberculous lesions are very rarely found elsewhere in the body, with the exception of the larynx.

Detection of Silicosis during Life

A detailed account of the signs and symptoms which characterize the various forms of silicosis need not be given here; it will suffice to outline the more important of them.

The most satisfactory diagnosis is that which is arrived at when the worker, as is usually the case in Johannesburg, has been under systematic observation for a long period; during this period, records of his radiographic appearances, weight, auscultatory signs, and circumstances of employment will have been made at intervals of about six months. In addition to this, bacteriologic examinations of the sputum are undertaken whenever any suspicion arises of his having become overtly tuberculous.

It has been found of considerable service simultaneously to record the radiographic appearances, the weight, and the nature of the employment upon a chart capable of displaying the records in chronologic order for ten years. The ordinates of this graph represent a scale of radiographic estimates of the amount of connective tissue throughout the lungs; the abscissas represent years. Records of weight are charted in the upper part of the scale, and in this region a line showing the standard weight, year by year, for an individual of the same height and age, is also traced. The nature of the occupation, and the results of sputum and blood tests, are recorded at the bottom of the graph in the vertical spaces between the abscissas. A specimen of this combined chart can be supplied to anyone practically interested in the subject of pneumonokoniosis.

Simple Silicosis

As a rule, the onset of simple silicosis is not associated with any marked fall in the line of weight; in fact this may rise considerably when the condition has become fully established. On the other hand, the line of weight will show depression in proportion to the activity of a tuberculous factor in the pulmonary condition, and this fall will often precede the appearance of suggestive radiographic signs.

The combined chart is specially serviceable in the routine medical supervision of the miners, since it usually gives a warning—often very marked—of the impending development of either silicosis or pulmonary tuberculosis.

The only signs of value which can

be elicited by ordinary clinical examination in cases of simple silicosis are general diminution of air entry, with a thin, high-pitched inspiratory murmur. Associated with this is some loss of elasticity of the chest wall, and, in cases of long duration, a diminished range of expansion; markedly reduced expansion is, however, usually suggestive of the presence of an active tuberculous complication.

The observations to be made from *a technically satisfactory* radiograph are of paramount importance. Apart from the assistance thus derived, it is never safe, even for the most experienced clinician, to venture a positive opinion as to the presence or absence of any form of this disease. The liability to error which exists when this method of examination has been omitted, or when the radiographic technic is poor, is well illustrated by the following. During last year, alone, the Medical Bureau examined 541 men who had been certified to be silicotic and who had been compensated, prior to the establishing of the Bureau in 1916. The certificates had been given by competent physicians of long experience on the Rand, who, however, had not the advantage of modern radiographic facilities. The Bureau found that in 225 (41 per cent.) of these compensated individuals there were no radiographic evidences either of silicosis or of tuberculosis. Such an experience is a striking commentary upon the practice of basing a diagnosis merely upon industrial history and auscultatory signs.

The radiographic appearances which characterize simple silicosis are usually first detectable as a gradual increase of the shadows normally cast by the connective tissue of the lungs. This increase is first noticed in the dendritic shadow in each lung, after which small-meshed reticular shadows appear, or become more pronounced, throughout both pulmonary areas. This physical sign (for I think the time has now come for including radiographic appearances among physical signs) is not specific to simple silicosis alone, but it is nevertheless developed over a period of two or three years before the appearance of the pathognomonic radiographic sign of that disease. This latter sign appears suddenly and develops to its maximum somewhat rapidly, *i.e.*, within a few months. It consists of a uniform and symmetrical mottling of the pulmonary areas, the mottling varying in texture from fine ("miliary") to coarse ("dappled") in different cases. The closeness of the individual spots of shadow varies directly with the amount of silica dust incarcerated in the lung, or, more correctly, with the number of nodular lesions to which the dust has given rise. Perhaps it should be remarked that the radiographic shadows are not produced by the silica particles themselves, but by the aggregations of connective tissue elements engendered by the presence of these particles.

With the lapse of time the individual shadows which compose the mottling become larger, less numerous, and less closely aggregated. The shadow of the heart also tends to change its shape and to become of the so-called "asthenic" or "vertical" type; the change in outline is from that of a curvilinear right-angled triangle toward that of a narrow, vertically placed rhomboid. This change in the

outline of the heart shadow is encountered in cachexias of various origin and is highly characteristic of pulmonary tuberculosis; it is nevertheless frequently found in well-developed cases of simple silicosis which are unassociated with any *obvious* indications of a tuberculous complication. It is significant of cardiac atrophy from any cause.

A peculiarity which is specially noteworthy from the clinical standpoint is that simple silicosis usually develops without giving rise to any symptoms which attract the attention of the patient. Even when the condition is fully declared, the only complaint is one of some shortness of breath on exertion. As previously stated, there may be a marked increase in body weight, and, as a rule, the individual has every appearance of good health. Cough, expectoration, pleuritic pains, loss of weight, and impaired vigor are usually indicative of the presence of overt tuberculous complication.

Tuberculosis with Silicosis

The condition of tuberculosis with silicosis is diagnosed primarily by the radiograph, assistance being derived from a knowledge of previous records, from the results of sputum examinations, and from a careful inspection of the combined chart of radiographic appearances and weight.

In tuberculo-silicosis, the radiograph shows that the hitherto uniformly mottled pulmonary areas are now marked by the addition of one or more unsymmetrical areas of shadow.

In silico-tuberculosis, the radiograph will have first disclosed an area of shadow (usually situated toward the apex, or extending from the hilus), while the remainder of the field, except for the probable presence of unsymmetrical peribronchial thickening, has been of normal appearance. At this time a diagnosis of simple tuberculosis will probably have been arrived at. Around the area of shadow, and extending to a variable degree over the pulmonary area on that side, a mottling now appears, usually within six months. This mottling is similar to but often coarser and less uniformly distributed than that which marks the entire pulmonary areas in simple silicosis. In workers who have inhaled only a slight excess of silica dust no definite mottling may develop around the main area of shadow, and the diagnosis during life must remain as simple tuberculosis; the postmortem discovery of the fact that the tuberculous trouble originated in an old, pigmented, and infected silicotic nodule will justify reclassification of the case under the heading silico-tuberculosis.

Differential Diagnosis

The uniform and symmetrical mottling which radiographically characterizes simple silicosis, and the unsymmetrical mottling of tuberculosis with silicosis, have hardly ever been found to be due to any other diseases. In one instance, the appearances of a simple silicosis of long standing were somewhat simulated by extensive, primary carcinomatosis of both lungs; while on two or three occasions an almost generalized coarse mottling has been found to be due to simple tuberculosis.

Tertiary syphilis has been recorded, in twenty-one instances, as having been responsible for the radiographic sign of an unsymmetrical peribronchial thickening, and the appearance, in many of the cases, of an opaque mass of irregular outline (usually having flame-shaped projections) in some part of one of the lungs. Antisyphilitic treatment has produced complete abolition of these radiographic signs in four of the cases, a marked improvement in ten, and no appreciable change in seven. In this connection it may be remarked that it has now become the custom of the Bureau to submit all cases of suspected simple tuberculosis, or tuberculosis with silicosis, to the Wassermann test of the blood. Of 568 such cases, 115 have given a positive reaction, but, as implied above, it has been in only a very small proportion of these that the pulmonary lesions were due solely to syphilitic infection.

Detection of Silicosis after Death

Dating from the Miners' Phthisis Act of 1912, the dependents of any miner who died of silicosis have received compensation, together with a substantial contribution toward the medical and burial expenses of the deceased. The Act of 1916 extended these "benefits" to cases in which silicosis, though not itself the primary cause of death, "was a contributing or predisposing factor to the cause of death;" while the Act of 1925 provides compensation if the mere existence of silicosis is discovered after death, whether or not such silicosis shared in causing the death.

As a fortunate consequence of these provisions, resort to postmortem examination has been almost the rule, and has enabled the Medical Bureau to acquire great familiarity with the changes in the lungs which are attributable to this disease. A detailed description of these changes will be found in the Annual Report of the Bureau for 1923 to 1924.

Under a previous heading in the present article[3] it was remarked that a few simply silicotic nodules may be found in the lungs (of miners who have neither suffered nor died from silicosis), and that minute simply silicotic lesions are usually discoverable in people who are not gold miners; it has therefore obviously been necessary for the Medical Bureau to adopt definite criteria as to what are the characters, and what the number, of silicotic lesions discovered postmortem which will justify the conclusion that the disease known as silicosis is present. These criteria are: (1) The lesions must be specifically those of silicosis, and must be both visible to the unaided eye and palpable to touch. (It will thus be seen that, in the considered opinion of the Bureau, microscopic silicotic changes, by themselves, do not constitute a disease.) (2) The lesions must be present in such numbers that, on the average, at least one will appear in each 5-cm. squared of the divided lung substance; provided that silicosis shall be deemed to be present when the visible and palpable lesions which have formed the starting point of a fatal silico-tuberculosis are limited in number, or even single.

In other words, the Medical Bureau holds that a few simply silicotic lesions do not constitute silicosis; but it nevertheless certifies that tuberculosis

[3] See under "Clinical Forms of Silicosis," p. 114.

with silicosis (in the form of silico-tuberculosis) was the cause of death when even a single infected lesion, of specifically silicotic character, has formed the primary site of a fatal pulmonary tuberculosis.

It may be remarked here that the lesions which, in the living man, constitute simple silicosis are very rarely discovered postmortem, and then only in men who have died from some cause other than silicosis; for, when this disease causes death, it does so because it has assumed the form of tuberculosis with silicosis.

TABLE 2.—DURATION OF PREVIOUS MINING WORK (EFFECTIVE PERIOD) IN EUROPEAN MINERS WHO HAVE DEVELOPED SIMPLE SILICOSIS SOLELY FROM EMPLOYMENT IN THE GOLD MINES OF THE RAND

	1917–1918	1918–1919	1919–1920	1920–1921	1921–1922	1922–1923	1923–1924
Total new cases among solely Transvaal miners.....	102	205	272	178	194	204	247
Effective period:	*mos.*	*mos.*	*mos.*	*mos.*	*mos.*	*mos.*	*mos.*
Shortest.....	22	30	36	39	39	54	52
Longest.....	216	246	270	226	232	301	265
Commonest.....	96–108	96–108	108–120	96–108	108–120	96–108	120–132
Average.....	109 (±2.15)	110 (±1.7)	114 (±1.5)	109 (±1.57)	115 (±1.78)	113 (±1.47)	123 (±1.59)

Effective Period

The total of all periods of actual employment in underground work, prior to the first appearance of silicosis in any particular case, is known to the Medical Bureau as the "effective period" for that case. In consequence of the compulsory examination of all working miners at intervals of not more than six months, the Medical Bureau is able to ascertain, with a considerable degree of accuracy, the time of first appearance of the specific radiographic signs of the disease, and can thus obtain a close approximation to the effective period. The Bureau began its work in August, 1916, and in each year since that date a substantial number of cases of the disease have developed in miners whose sole work has been in the gold mines of the Transvaal. Table 2 supplies information as to the effective periods (stated as months of actual employment) for all cases of simple silicosis which have originated in each year among such men.

It will be seen that, if a miner contracts simple silicosis solely from work in the mines of the Rand, the shortest time in which he now does so is about four and a quarter years; and that, for all such miners as contract the disease, the average time is now about ten and a quarter years. That the average effective period has become definitely lengthened of late years may be reasonably attributed to the greatly improved conditions underground with respect to dust. Incidentally, it may

be remarked that this observation appears to justify the following conclusion: that the length of the effective period commonly prevailing in any silicosis producing industry will be found to be inversely proportional to the intensity of exposure to silica dust.

Incidence

It was remarked in the preceding section that the average duration of underground work in the Transvaal, prior to the development of simple silicosis, is now about ten and a quarter years; the mistake must not be made, of course, of supposing that all, or even the majority, of the miners who have worked for ten and a quarter years will develop the disease; it is only about 3 per cent. of the total working miners, and only about 7 per cent. of those who have worked for ten years or longer, who annually become silicotic in any form.

Working Miners

The relative numbers, per hundred thousand of the working miners, of those who have annually developed simple silicosis is shown in Table 3, and this is also utilized for recording the annual incidence of the two other legally recognized diseases. Since miners with simple silicosis are not compelled to leave their work, there are always a greater number of them in employment during any one year than have become silicotic in that year; in other words, the annual prevalence rate for this condition is greater than the production rate, as is shown in the table. If, however, simple silicosis in a working miner becomes complicated by overt tuberculosis (tuberculo-silicosis), or if a previously non-silicotic miner develops either silico-tuberculosis or simple tuberculosis, immediate retirement from mining work is compulsory; the production rates for tuberculosis with silicosis and for simple tuberculosis are therefore practically identical with their prevalence rates, and the latter are accordingly omitted from the table.

The most satisfactory feature of Table 3 is the marked decline in the incidence both of tuberculosis with silicosis (which is the gravest form of the three diseases) and of simple tuberculosis. The decline in the former is not due merely to the more frequent retirement of simply silicotic miners before they become tuberculo-silicotic, for the production rate for silico-tuberculosis has also fallen, from 338, in 1918 to 1919, to 131, in 1923 to 1924.

The unusual height of the prevalence rate for simple silicosis in 1919 to 1920 was partly due to the first operation of a provision of the 1919 Act, which required the recognition of a new stage in the disease. This was called the "ante-primary stage," and is constituted by "the earliest detectable specific physical signs" of the disease. The increase was also contributed to by the return of large numbers of miners from military service; many of these, as remarked under the section "Latent Silicosis,"[4] had acquired a liability to develop the disease (upon the advent of a tuberculous infection) before they left their work.

A tendency for the production rate for simple silicosis to rise is manifest in the last three years recorded in the

[4] See p. 113.

table, and returns available at the time of writing show that this tendency

TABLE 3.—INCIDENCE OF COMPENSABLE LUNG DISEASES UPON THE WORKING MINERS

(Rates per 100,000)

	1916–1917	1917–1918	1918–1919	1919–1920	1920–1921	1921–1922	1922–1923	1923–1924
Number of working miners examined	14,625	15,066	15,074	15,468	13,682	13,457	12,691	12,159
Simple silicosis:								
Prevalence	5,046	4,646	4,670	8,869	3,273	3,224	3,198	3,716
Production	....	1,179	2,538	2,787	1,756	1,950	2,148	2,652
Tuberculosis with silicosis:								
Production	....	860	852	163	124	178	149	164
Simple tuberculosis:								
Production	....	259	255	265	322	163	196	90

is being maintained. This is the most embarrassing feature of the position as it is today. That this increased production of the disease is not due to any increased dustiness of the air in the working places is quite evident from the reports of the officials who continually supervise these conditions. It should, moreover, always be remembered that, although the present incidence of silicosis upon an industrial community may be immediately determined by the present existence of sources of tuberculous infection, the prerequisite condition of latent silicosis is, as it were, a composite reflection of many years' exposure to dust. It is also worthy of special remark that practically all the cases of silicosis (in any form) which have originated since 1916 have been in men who were working as miners before that year, and who had not, therefore, been required to pass the initial examination of the Medical Bureau; among more than 7,600 European men who have started mining since 1916, after passing the initial examination, there have been, up to the time of writing, only nine cases of silicosis in any form.

The question naturally arises: If the increased production of simple silicosis is not due to increased dustiness, what is the cause? There are two probable explanations: (1) The number of long-service miners (and therefore the number of those in the condition of latent silicosis) has steadily increased; and (2) these miners have been more frequently exposed to the determining influence of a tuberculous infection. In the year 1918 to 1919, only about 30 per cent. of the working miners had been in underground occupation for longer than six years; but by the year 1923 to 1924 this proportion had risen to 60 per cent. This tendency for the long-service miners to remain in employment has also become evident among the native laborers; more than 1,000 experienced natives, of longer than five years' continuous underground

service, are now at work. Unfortunately, this retention of the services of experienced natives has been secured without the safeguard of the periodic radiographic examination to which all European miners are subjected. The result has been that many natives have been carelessly retained for underground work after they have become tuberculous, and they have thus increased the sources of infection to which the miners are exposed.

Native Laborers

No account of silicosis on the Rand would be complete which did not afford some information as to its incidence upon the native workers, and a table, similar to that for the miners, is therefore provided (Table 4). On comparing these two tables it will be immediately seen that the two classes of worker are very differently affected by simple silicosis and by simple tuberculosis. In the year 1923 to 1924, for example, simple silicosis was relatively fifty-six times more frequent among the miners, while simple tuberculosis was about three and a half times more prevalent among the native laborers. Tuberculosis with silicosis (which usually appears in our native laborers as silico-tuberculosis) is about equally prevalent in the two classes.

The relative rarity of simple silicosis among the natives is merely the result of the prevailing shortness of their periods of employment; the European miner is more or less in permanent employment, whereas the native usually works for only six or nine months at a time, returning to his kraal for a year, and often longer, between his spells of mining work. Nevertheless, about 95 per cent. of the native laborers at present employed have worked in the mines on previous occasions, and it is significant that almost exactly the same percentage of them give a positive result to the von-Pirquet test.

It will be noticed from the table that the annual prevalence of ordinary pulmonary tuberculosis has undergone very definite reduction, but that this reduction is, relatively, not nearly so marked as among the European miners. The rates refer, of course, to the whole body of native laborers and, from this point of view, they do not reflect an incidence of the disease much in excess of that among the adult males of some of the industrial towns of Great Britain. In connection with the question of the exposure of the European miners to special risks of infection, however, it is very significant that, in 1923 to 1924, the prevalence rate for this disease among the long-service underground natives, previously referred to, was 1,614—nearly five times the general rate.

This is a convenient opportunity to remark that simple tuberculosis commonly appears in the South African native as pneumonic phthisis, or follows the "infantile" type of the disease—acute, with marked tendency to generalization, and terminating fatally within two or three months. Examples of "monkey" tuberculosis are also frequently encountered, characterized by great enlargement of the abdominal lymph nodes and the appearance of large, white, necrotic masses in the spleen and liver.

Rates of production are not given in the table, because the Medical Bureau does not examine and radio-

graph all the native laborers at regular intervals, as in the case of the European miners; exact knowledge as to when each case originates is therefore wanting. The medical supervision of the natives is undertaken by privately appointed mine medical officers, who send forward to the Bureau any native whom they suspect of having become the subject of a compensable pulmonary disease. When it is remembered that each mine medical officer is in charge of, on the average, about 6,000 natives, and that some are responsible for as many as 12,000, it will be appreciated that an individual examination of every native at intervals of three months is a physical impossibility. The Miners' Phthisis Act, in recognition of this impossibility, ordains that the weight of every native laborer shall be taken and recorded every three months, and a special examination made of all those who have lost weight. Experience has shown, however, that, for one reason or another, this system does not lead to the detection of more than about 50 per cent. of the cases of simple tuberculosis and of tuberculosis with silicosis which are finally discovered. The result is that many tuberculous natives are not detected until they have become too ill to work; by which time they will probably have communicated their infection to some of their fellow workers, including the European miners.

TABLE 4.—INCIDENCE OF COMPENSABLE LUNG DISEASES UPON THE NATIVE LABORERS

(Rates per 100,000)

	1916–1917	1917–1918	1918–1919	1919–1920	1920–1921	1921–1922	1922–1923	1923–1924
Average number of native laborers	191,202	180,415	171,959	178,571	168,547	160,600	179,182	178,006
Simple silicosis:								
Prevalence	47	17	27	70	79	27	41	66
Tuberculosis with silicosis:								
Prevalence	131	128	219	220	163	126	182	175
Simple tuberculosis:								
Prevalence	576	524	458	444	480	361	370	328

In order to mitigate the mischief of which the overlooked native "carrier" of tuberculous infection is the source, I have lately proposed utilizing, on a large scale, X-ray examination by means of the fluorescent screen. This method is now undergoing a preliminary trial. With the observer seated in an X-ray proof cabinet, the individuals pass in procession and press the front of the chest against the screen, which forms part of the wall of the cabinet. Any marked pulmonary damage is instantaneously recognized.

Constitutional and Racial Susceptibility to Silicosis

It is very doubtful whether the development of silicosis is markedly predisposed to by age, race, or any

individual peculiarity—other than a racial susceptibility to tuberculosis, or the presence in the individual of a latent tuberculous infection; apart from these exceptions, the incidence of the disease appears to be dependent upon only two factors—namely, the duration and intensity of exposure to silica dust, and the frequency and intensity of exposure to tuberculous infection.

It was formerly supposed that the fact that silicosis commonly declares itself between the ages of 39 and 42 years indicated the existence of a special liability to the disease at this period of life; statistical investigation has shown, however, that the significant influence is the duration and nature of the work underground, rather than the age. It is of interest to note that, while the average age at which simple silicosis has most frequently declared itself among the European miners has varied from 39 to 42 years, in the different annual batches, the average age for the occurrence of simple tuberculosis has been from 36 to 41 years.

The remarkably slight incidence of simple silicosis upon the native laborers has also been considered as suggestive of some degree of racial insusceptibility to the disease; but the true explanation of the phenomenon is to be found in the fact, already stated, that their periods of work in the mines are, in the great majority of instances, relatively very short. A detailed investigation has recently been made into the condition of all those natives (about 1,000 in number, and constituting only about 0.57 per cent. of the total native labor force) who have been in continuous underground employment for five years or longer; the results showed that, when engaged in similar work for similar periods, the liability of the native and the European to develop silicosis had been practically the same. The validity of this conclusion is also supported by animal experimentation, for simple silicosis can be produced with equal ease in various kinds of small animals when they are exposed for the same periods to similar concentrations of silica dust.

There has long been a theoretical prejudice, and it still prevails, that habitual mouth-breathers will contract silicosis more readily than those who inspire through the nose; if this be legitimate, the special liability is probably the consequence of associated tuberculous damage in the lungs, rather than of the more ready access of dust to these organs. The extensive experience of the Medical Bureau has nevertheless neither confirmed nor disproved this supposed susceptibility of mouth-breathers, doubtless because such people are but very rarely found among the miners, and they have not been considered as physically fit to pass the initial examination.

A very definite susceptibility to the disease, especially in the form of silico-tuberculosis, is present in individuals of any race in whom previous tuberculous infections have produced the slightest pulmonary damage, even although the infective process has become completely latent. The radiographic signs of peribronchial thickening (especially if it is unsymmetrical), and of the asthenic heart (which is almost always accompanied by deficiency in weight), are very definite warnings that the individual,

if he engages in underground work, will develop either simple tuberculosis or silico-tuberculosis. The intuitive disapprobation of candidates for mine employment who present the scars of cervical adenitis, who are deep-eyed, underweight, and elderly, or who come from certain places—such as Madeira—is doubtless an unconscious recognition of the probable presence in such people of latent pulmonary damage of tuberculous origin.

Special Hazard of Certain Underground Occupations

In consequence of the prevailing practice of changing from one class of work to another, there is but a very small proportion of the miners who have always followed only one class of occupation. For this reason it is difficult to allocate to each class of work its relative responsibility in the production of silicosis. There is, however, one occupation which is demonstrably more hazardous, in the matter of producing silicosis, than any other, and this is machine drilling. The average effective period for all those who have become silicotic in each year, from work with machine drills only, has always been markedly shorter (by from ten months to twenty-five months) than the average effective period for the other new cases in the same year.

The explanation of this special liability of the machine driller is obvious. He is a producer of dust, and he works nearer to the site of its production than anyone else; moreover, if he is engaged in "development," he is usually stationed in those places (dead ends) which are the most difficult to ventilate efficiently.

Stages of Silicosis

The stages into which the course of silicosis may be divided differ according as we view the disease from its clinical or from its legal aspect.

From the Clinical Aspect

When discussing "Clinical Forms of Silicosis" it was stated that the condition of simple silicosis is not accompanied by any marked degree of invalidism[5]: if, and when, the condition passes into that of tuberculo-silicosis, however, an illness is established which usually runs a course very similar to that of ordinary bronchopneumonic pulmonary tuberculosis, excepting that it advances to a fatal termination with much greater certainty and rapidity. When the disease has originated in the form of silico-tuberculosis, a breakdown in health is immediately evident, and the decline is even more rapid than with tuberculo-silicosis. These facts lead to the conclusion that, from the clinical point of view, silicosis cannot reasonably be held to have more than two stages—simple silicosis and silicosis accompanied by overt tuberculosis—and that the second stage need not necessarily have been preceded by the first stage.

If called upon to assess the capacity for work of a silicotic, the Medical Bureau simply records its opinion under one of the four following headings: (1) "ordinary work;" (2) "moderate work;" (3) "light work;" (4) "no work at all."

From the Legal Aspect

Previous to the passing of the 1919 Act, the law in South Africa held the

[5] See p. 114.

employer financially responsible for the occurrence of silicosis only if the disease had impaired the wage earning capacity, or had caused, or assisted to cause, death. The enactment of 1919 enlarged the employer's responsibility so as to cover the mere presence of the disease during life, irrespective of whether it had been detrimental to the capacity of the worker; while the latest law (1925) prescribes a similarly unconditional award to the dependents if the disease is discovered by postmortem examination.

Under the present law silicosis is divided into three stages, which are named and defined as follows:

(a) in the *Ante-primary Stage*, when it is found by the Bureau that the earliest detectable specific physical signs of Silicosis are or have been present; whether or not capacity for work is or has been impaired by such Silicosis;

(b) in the *Primary Stage*, when it is found by the Bureau that definite and specific physical signs of Silicosis are or have been present, and that capacity for work is or has been impaired by that disease, though not seriously and permanently;

(c) in the *Secondary Stage*, when it is found by the Bureau that definite and specific physical signs of Silicosis are or have been present, and that capacity for work is or has been seriously and permanently impaired by that disease, or when it is found by the Bureau that Tuberculosis-with-silicosis is or has been present.

It may be remarked that the words "has been" and "have been" in the foregoing definitions do not imply recovery from the disease; they relate to posthumous claims by dependents.

Prognosis

A reasonably accurate estimate of the expectation of life in a case of silicosis is of importance to the efficient management of the Compensation Fund; and it is, of course, of supreme moment to the affected individual. With the establishing of the Medical Bureau it became possible systematically to collect the requisite data, and Table 5 records the information which has so far become available. It represents the case mortality, in each year succeeding the year of onset, of the various forms of compensable lung disease among the European miners, over a period of seven years. Deaths from causes entirely independent of the pulmonary disease have been excluded, but it is of interest to note the ratio which they have borne, during the whole period, to the deaths from the lung diseases: In each thousand deaths of cases originating as simple silicosis there have been 170 deaths from other causes; in each thousand deaths of cases of tuberculosis with silicosis, and of simple tuberculosis, there have been nineteen and twenty-five deaths, respectively, which were due to other causes.

It will be seen (Table 5) that, for each year following that in which the disease first declared itself, the deaths per thousand of the surviving cases are given in column A, and the deaths per thousand of the original total of cases in column B. The former provides some measure of the probability that any particular surviving patient may die in any particular year; while the latter suggests what the mortality among the original number of cases will have amounted to by the end of any particular year.

The rates in column B, under the different headings but especially under "Simple Tuberculosis," show some

irregularity of progression; this is mainly because the total number of cases available for tracing over the longer periods, as shown in parenthesis after each rate, tends to become inefficiently small.

The question of forecasting the probability that a case of simple silicosis will become overtly tuberculous has been considered in a former section; it may now be remarked that the prospects of life when this change has occurred are here indicated under the heading "Tuberculo-Silicosis."

Perhaps the most important disclosure made by this table is the marked difference in the fatality of the diseases. It will be noticed that, by the end of the seventh year, 29 per cent. of cases originating as simple silicosis, 46 per cent. of cases of simple tuberculosis, 86 per cent. of cases of tuberculo-silicosis, and 89 per cent. of cases of silico-tuberculosis, have died of their disease.

Efforts to Prevent Silicosis

It is entirely wrong, in my opinion, to assume that gold mining cannot be carried on without giving rise to silicosis in the workers. If such an assumption were true, the question whether the industry should not be totally prohibited would immediately arise, for the continuance of any commercial enterprise which *necessarily* involves the sacrifice of even one human life is indefensible. A sane and educated person would not maintain that railway transport should be interdicted because many lives are annually lost upon the railways. This is because it is recognized that railway accidents are essentially preventable

TABLE 5.—FATALITY IN EACH YEAR FOLLOWING THE YEAR IN WHICH THE DISEASE FIRST APPEARED IN EUROPEAN MINERS

(A) = rate per 1,000 of cases surviving at beginning of each successive year; (B) = rate per 1,000 of original cases (cumulative year by year)
(The number of cases from which the case mortality is deduced is given in parenthesis after each rate)

SUBSEQUENT YEARS	SIMPLE SILICOSIS		TUBERCULOSIS WITH SILICOSIS				SIMPLE TUBERCULOSIS			
			Tuberculo-Silicosis		Silico-Tuberculosis		Open		Closed	
	(A)	(B)	(A)	(B)	(A)	(B)	(A)	(B)	(A)	(B)
1st	24 (1,971)	24 (1,971)	301 (169)	301 (169)	421 (171)	421 (171)	180 (155)	180 (155)	87 (57)	87 (57)
2nd	30 (1,585)	55 (1,652)	286 (115)	503 (165)	333 (87)	625 (155)	169 (118)	324 (145)	117 (51)	196 (56)
3rd	34 (1,283)	92 (1,395)	291 (79)	644 (163)	176 (51)	695 (138)	102 (88)	382 (128)	52 (38)	250 (48)
4th	48 (998)	139 (1,136)	163 (55)	694 (157)	189 (37)	750 (120)	43 (69)	421 (114)	32 (31)	250 (40)
5th	44 (736)	191 (900)	173 (46)	741 (155)	115 (26)	780 (105)	37 (54)	393 (89)	58 (17)	238 (21)
6th	63 (380)	262 (502)	228 (35)	797 (148)	272 (22)	818 (88)	48 (41)	422 (71)	... .	... .
7th	55 (109)	294 (153)	166 (12)	860 (79)	0 (4)	891 (37)	0 (19)	457 (35)	... .	... .

occurrences; the more intelligently they have been investigated and provided against, the less frequent have they become. Investigation into silicosis has similarly disclosed the fact that it is essentially a preventable disease, but that, unlike railway accidents, the results of negligence are not revealed until after the lapse of many years.

The employers upon the Rand have encouraged and liberally supported researches into the nature, causation, and prevention of silicosis. This has been done, not only in acknowledgment of the moral obligation, but also because the law has imposed a heavy financial responsibility upon them in the matter of compensation for the occurrence of this disease and its common associate, pulmonary tuberculosis.

The measures which have been taken to prevent the occurrence of silicosis and tuberculosis may be roughly divided into: (1) those of a mechanical nature, and (2) those of a medical nature.

Mechanical Precautions

Schedule XIX of the Report upon the work of the Miners' Phthisis Medical Bureau for the year 1918 to 1919 contains a historical summary of the mechanical precautions which had been adopted up to that date. Practically all these precautions, as well as those more recently introduced, have been directed against the creation of dust *during work*, and toward the supplying of the workers with air which is free from dust and the fumes of explosives. A detailed discussion of these precautionary measures would not be appropriate to the present occasion; probably the most beneficial of them have been as follows:

Prohibition of machine drills unless provided with an axial water feed;

Prohibition of the blasting of the "cut and round," separately, in the same shift;

Providing that blasting shall take place only once in twenty-four hours, and always at the end of the shift;

Prohibition of working in, or entering, any place the air of which contains dust or fumes detectable by the unaided senses;

Securing that an adequate supply of clean air, and of water for dust prevention, shall be furnished to all working places;

Providing that no broken rock shall be moved unless thoroughly wetted.

The duty of enforcing these, and all similarly mechanical precautions, is carried out by officials of the Department of the Government Mining Engineer.

Medical Precautions

The precautionary measures of a medical nature fall within the province of the Medical Bureau, but before these measures are detailed a few words may be said about the essential characters of the Bureau. The Medical Bureau administers the medical sections of the Miners' Phthisis Act according to the Regulations issued under that Act. The Bureau is conducted at the sole expense of the government. The members and other officers of the Bureau are appointed by, and are solely responsible to, the government. The members of the Bureau are medical men who are not permitted to engage in private practice, and who are required to devote their whole time to the work of the Bureau. The certificates issued from

the Bureau represent either the unanimous or the majority opinion of its members; they never represent the opinion of only one or two. The Bureau assembles in full session, at the close of every day's work, to consider and adjudicate upon the cases which have been examined during the day.

The medical measures which are of a precautionary or preventive nature are as follows:

A person who is not a miner may not go underground for any purpose unless he has been examined by the Medical Bureau (the "Special Examination") and certified not to be suffering from tuberculosis.

No man may become a miner until he has been certified by the Medical Bureau (after an "Initial Examination") to be free from disease of the lungs, and physically fit in other respects to follow the occupation. The Bureau considers that the scrutiny of a technically satisfactory radiograph of the chest forms an essential part of both the "Special" and the "Initial" examinations.

Every man who is employed in underground work must be examined, at a "Periodical Examination," at least once in every six months; this examination includes the taking of a radiograph. If he is found to have developed simple silicosis, he is informed of the fact, by confidential letter, and is at liberty to take his "compensation" and retire from the work. If, however, he is found to have developed either simple tuberculosis or tuberculosis with silicosis, he is prohibited from again going underground, and must take his "compensation" forthwith.

The discovery at a "Periodical Examination" of any disease calling for medical treatment—such as syphilis, valvular disease, etc.—or of a condition considered premonitory of tuberculous disease, is confidentially intimated to the miner, and he is advised to consult his own doctor about it.

Laboratory examinations of sputum, urine, blood, etc., are undertaken as often as deemed necessary, and without any restriction as to number.

In all cases of the suspected development of tuberculosis, the miner is admitted to the Observation Ward of the Bureau, where he remains under close medical supervision for three days.

No miner is finally certified to be the subject of a compensable lung disease until the reaction of his blood to the Wassermann test has been ascertained; and, if this be positive, the results of specific treatment may be awaited in certain cases.

All the examinations of the Medical Bureau are carried out free of cost to the examinees, and when an employed miner attends for his "Periodical Examination" his day's wage is guaranteed by his employer.

If the Bureau is apprehensive lest a miner be developing a disease of the lungs, he is examined again before the expiry of the usual period of six months.

When genuine doubt exists as to the condition of a miner, and this doubt cannot be resolved by further examinations, it is the custom of the Bureau to give its decision in favor of the miner.

The Medical Bureau works in close cooperation with the South African Institute for Medical Research; it is accommodated in the same buildings, and is thus enabled to utilize the laboratory services, library, museum, and postmortem laboratory of that institution.

Changes in Character and Incidence of Compensable Lung Diseases

The Bureau has obtained quite definite evidence of the benefits which have resulted from the adoption of the measures outlined in the preceding section. Both the mechanical and the medical precautions have fully justified themselves, although it is not always possible to award the sole credit for a particular improvement to either one or the other class of precautionary measures. Indications of progress have been referred to in certain of the previous sections, but we

may now enumerate them more fully.

Improvement in General Standard of Health.—It is to be noted, in the first place, that there has been a marked improvement in the general health of the miners. This has been noticed and commented upon by several of the mine managers who have had long experience on the Rand. Some indication of this advance is afforded by the fact that, whereas in the year 1917 to 1918 the Bureau confidentially advised 323 miners of the existence of some disease or menace to their health, only sixty-eight required to be similarly advised in 1923 to 1924.

Effect of Initial Examination.—The justification for the various precautions and prejudices exercised by the Medical Bureau at its initial examination (as outlined under the section "Constitutional and Racial Susceptibility"[6]) is shown by the incidence of the compensable diseases upon the Europeans who have passed this examination. The total incidence of these diseases upon such men since 1916 is recorded in Table 6.

Of the seven men who developed silicosis, five had followed the occupation of miner before passing the initial examination.

The criticism has often been heard that the standard of initial physical fitness set by the Bureau is unnecessarily high, and that the stringency of the examination therefore prevents many workers from obtaining employment. Such criticisms are not defensible, for the only feature of physical fitness which is specially stressed is that of the pulmonary condition, and this has been done solely in the best interests of the workers themselves. It is also to be noted that, although the Medical Bureau has found it necessary to reject, on the average, about 60 per cent. of the candidates, it has nevertheless passed more than twice as many as are able to secure mining work.

Reduction in Incidence of All Forms of Pulmonary Tuberculosis.—In the whole body of working miners the attack rate for simple tuberculosis has been reduced from 259 per hundred thousand, in 1917 to 1918, to 90, in 1923 to 1924; while the incidence rate for tuberculosis with silicosis has fallen in the same period from 860 to 164 (Table 3). Among the native laborers the incidence rate for simple tuberculosis has fallen from 576, in 1916 to 1917, to 328, in 1923 to 1924 (Table 4).

Appearance of Simple Silicosis in Less Severe Form.—The production of cases of simple silicosis in the "ante-primary stage" was first recorded in the year 1920 to 1921, for this stage was not legally recognized until August, 1919. In the former year, in addition to the ante-primary stage cases, the disease declared itself immediately in the "primary stage" in seventeen instances; while during the year 1923 to 1924 only one case originated in the primary stage. No case has originated directly in the "secondary stage" since the year 1918 to 1919.

Increased Prospects of Survival in Cases of Simple Silicosis.—That the disease is assuming a less severe type is indicated by the fact that the proportion surviving, year by year, out of each year's batch of new cases of simple silicosis is increasing; for example, after the lapse of four com-

[6] See p. 129.

plete years, 76.4 per cent. of the 1917 to 1918 cases, 81 per cent. of the 1918 to 1919 cases, 84.4 per cent. of the 1919 to 1920 cases, and 88.5 per cent. of the 1920 to 1921 cases were alive.

TABLE 6.—INCIDENCE OF COMPENSABLE LUNG DISEASES UPON MINERS WHO HAVE PASSED THE INITIAL EXAMINATION OF THE MEDICAL BUREAU

	1916–1917	1917–1918	1918–1919	1919–1920	1920–1921	1921–1922	1922–1923	1923–1924
Number of Europeans employed in underground work after passing the initial examination	621	1,737	2,741	3,592	4,111	4,798	5,512	6,132
Simple silicosis	0	0	0	1	0	2	3	0
Tuberculosis with silicosis	0	0	0	0	0	0	0	1
Simple tuberculosis	0	0	1	1	4	1	3	2

Lengthening of Effective Period.—In those instances (never less than 149 in each year) in which simple silicosis has developed in the ante-primary stage as a result of work on the Rand only, the duration of underground employment prior to the appearance of the first signs of the disease has become longer; thus, in 1920 to 1921, the average effective period for these cases was 112 months, the shortest period was 53 months, and the longest 122 months; in 1923 to 1924 the average period for such cases was 125 months, the shortest 60 months, and the longest 149 months.

Decrease in Extent and Number of Silicotic Lesions Found Postmortem.—The extent of pigmentation and the number of silicotic lesions which are found in the lungs of fatal cases of all forms of the disease depend upon the number of silica particles which have been incarcerated. A comparison of the pathologic records for the period 1916 to 1918 with those for the period 1922 to 1924 shows that the amount of pigmentation and the number of silicotic lesions, as generally encountered, have markedly decreased; in the former period, pigmentation was noted as being "in great excess" at 11 per cent., and silicotic lesions as being "abundant" at 13 per cent., of the necropsies; in the latter period, "great excess" of pigmentation was not disclosed in a single case, while in only one instance could the lesions be classed as "abundant." Another change is also significant. In the earlier years of the work of the Bureau, the pulmonary changes associated with ordinary bronchopneumonic tuberculosis were never seen in association with any specifically silicotic lesions; every part of the lung in which they might have appeared presumably contained a sufficient number of silica particles to insure that any development of tuberculous infection would assume the form of infective silicosis. During the last few years, however, specimens have frequently been encountered in which the ordinary

nonpigmented lesions of the common type of pulmonary tuberculosis existed side by side with a few silicotic lesions.

Other Diseases among Mine Workers

The relations between silicosis and tuberculosis are so intimate that it is not surprising to find that, of all diseases associated with silicosis, pulmonary tuberculosis is the most common. In 726 necropsies made between 1916 and 1924, on the bodies of European gold miners who had died from a variety of diseases, active tuberculous lesions were found in the lungs in 42.2 per cent. of the examinations; in 40 per cent. these lesions were accompanied by those of other pulmonary diseases, while in 2.2 per cent. tuberculosis alone was present.

Lobar pneumonia was formerly very rife among the native laborers, but it has been almost completely abolished by prophylactic inoculation. Despite the fact that the European miners are not inoculated against this disease, it is doubtful whether it is very markedly prevalent among them; in the 726 necropsies just referred to, lobar pneumonia was found to be present in 15.6 per cent., being associated with other pulmonary diseases in 8.6 per cent., and occurring alone in 7 per cent. of the examinations.

Rheumatism is very rarely encountered among the mine workers; this fact is specially noteworthy because the prevention of dust in the underground working places necessitates the continual and abundant use of water.

Bright's disease does not appear to be more frequent among the miners than among the general population.

Ankylostomiasis is occasionally found, but only among the native laborers. The strongly acid reaction of the water naturally present in most of the mines is inimical to the larvae of the worm.

Miners' nystagmus, so far as one's knowledge goes, has never originated in a worker either in the gold mines or in the coal mines of South Africa.

Primary cancer of the lung was discovered at four of the 726 necropsies on Europeans; the specific signs of silicosis coexisted in only one of these cases.

Future Progress

Exposure of the workers to dangerous dust has been enormously reduced of late years, as the result of systematic and sustained efforts. That the great majority of the cases of silicosis now appearing among the Europeans are in those who were subjected to the old conditions is a fact which suggests that their latent silicosis was mainly acquired before dust prevention measures had become general. When these measures were first organized on a large scale it was supposed that they, by themselves, would bring about the suppression of silicosis; but subsequent experience has shown that the second prerequisite factor in the disease, tuberculous infection, cannot be ignored.

Complete protection from dust has nevertheless not yet been secured, especially in shaft sinking and development, and a continuance of the special efforts in this direction is called for. Any further and markedly effective progress, however, is contingent upon a full and wide recogni-

tion of the communicability of tuberculosis, and of the necessity for taking active measures for protecting the mine worker from this infection. The most frequent sources of the infection to underground workers, at the present time, are the overlooked cases of silico-tuberculosis, and of simple tuberculosis, among the native laborers; these people often show no obvious signs, and make no complaint, of illness.

EXPERIMENTAL ELECTRIC SHOCK*

R. W. Ian Urquhart, M.A., M.B.

From the Laboratory of Physiology, University of Toronto, Toronto, Canada

Introduction

THIS research had its inception in the previous work of Mr. Wills Maclachlan, Consulting Engineer, Toronto, on the history and methods of resuscitation as applied to the victims of electric accidents. As a result of this work, it was evident that if any progress were to be made in this field it would be necessary to obtain further information as to the actual effect of the electric current on laboratory animals. Accordingly, at Mr. Maclachlan's suggestion moneys were obtained from the Hydro-Electric Power Commission of Ontario, to defray the expenses of an investigation into the causes of death from electric shock and, if possible, to learn if anything further could be done to aid in the resuscitation of its victims. Arrangements were therefore made with Prof. J. J. R. Macleod of the University of Toronto to carry out such an investigation in the Department of Physiology under his direction. Electrical facilities were supplied by the Hydro-Electric Power Commission of Ontario.

The startling and frequently fatal results observed when a person comes into contact with a charged electric conductor have excited the interest of the medical profession since 1879, when the first fatal accident occurred from a dynamic electric circuit. Many researches have been undertaken to discover, if possible, the cause of death in such cases and to formulate suitable remedial measures to protect those exposed to the possibility of such accidents. The men in the field, on the other hand, have been concerned mainly with the perfection of safety appliances and with the adoption of preventive regulations in order that these untoward accidents may be reduced to a minimum. The practice, now almost universal, of instructing the field worker in the recognized principles of resuscitation, has been the means of saving many lives. With the increasing use of electric energy in industry, however, the number of casualties continues to grow, owing either to the lack of immediate application of the now known principles of resuscitation or to fatality in spite of their efficient application. The necessity for further investigation into these problems becomes evident.

Review

Jex-Blake (1), in the Goulstonian Lectures of 1913, reviewed and summarized the whole field of electric shock up to that time. He quotes

*Received for publication Dec. 27, 1926.

largely from the outstanding work of Prevost (2) and Battelli (3). From his wide study of the reported experiments on animals and of the records and statistics of accidents to human beings, he reaches the following conclusions and applies them to man:

1. The existence of an idiosyncrasy to electric currents in man is doubtful.

2. Death in human beings may occur in different ways:

a. It may be brought about by a prolonged tetanus, causing death by asphyxiation;

b. By primary heart failure or ventricular fibrillation;

c. By failure of respiration, either through nerve inhibition or through actual damage to the central nervous system;

d. By delayed death, the result of burns or complications.

3. The electrical details are of great importance. With high voltages (over 500 volts) the brain and the spinal cord are more often affected, while with lower voltages the heart is the usual point of action.

4. Artificial respiration is recommended as the only method of treatment holding out any hope. (This may or may not be accompanied by so-called "counter shock," *i.e.*, stimulation of sensory nerves by pressure or blows.)

Apart from subsequent minor additions to our knowledge of the subject, these conclusions may properly be regarded as representing the present general conception of the problem. Reference must, however, be made to the work of Jellinek (4) who, by performing numerous experiments on animals and by collecting data from the reports of a large number of accidents, has contributed much to the literature on the subject. Jellinek stresses the importance of immediate and continued application of efficient artificial respiration, particularly when the "apparent death" is due to respiratory failure. He believes that if fibrillation of the heart is present, the slight massage afforded by this measure may have some beneficial effect on the patient.

Borruttau (quoted in (5)), in 1918, from a study of the records of fatal accidents in Germany, reported that 90 per cent. of the fatalities were due to ventricular fibrillation and suggested that continued artificial respiration is useless unless evidence is obtained that the circulation is intact.

As a result of his work on dogs, MacWilliam (6) also subscribes to the theory that ventricular fibrillation is the most important factor in electric fatalities.

Scott Ram (7) made an analysis of the records of electric accidents in England from 1912 to 1921. In the majority of the cases, artificial respiration had been attempted. He points out that of those who had received over 650 volts, 62.5 per cent. recovered, while only 39 per cent. of those who had received under 250 volts were saved. This gives some indication as to the determining factor in the fatality. The higher voltages have a somewhat selective action on the respiratory apparatus, and artificial respiration is frequently of value; while the lower voltages act mainly on the heart, causing an irremediable fibrillation. Provided proper treatment by artificial respiration is promptly applied, the risks to life are therefore decidedly less with relatively high voltages than with lower ones.

The available records of electric accidents in Ontario to some extent confirm these conclusions. A relatively higher percentage of recoveries is apparent and may be attributed: first, to the nature of the current—the majority of the power lines carrying high tension; and, secondly, to the organization and training of the electrical worker in the prone pressure method of artificial respiration. How further to increase the percentage of recoveries is the problem with which we are confronted.

An important fact, first observed by Prevost (2) and Battelli (3) and later confirmed by many other workers, is the decided difference in the effect of similar electric currents on animals of different species and, indeed, although in a less marked degree, on animals of the same species. Great care must therefore be taken in the interpretation of experimental evidence and in its application to man. This is particularly true in regard to observations on the heart. The marked difference in the susceptibility to ventricular fibrillation of the heart of the dog and of the rabbit is well known, whereas there does not seem to be the same variability in the reaction of the nervous system in these and other animals. The effect of the electric current on these two systems has, however, been clouded by the involvement of other parts of the body in the path of the current. It seemed to us important that the problem should be reinvestigated by making observations in which the electrodes were so placed that the nervous system and the cerebral nerve centers would alone be involved, and it is with an account of these investigations that the present report is concerned.

Present Investigation

Inasmuch as the rabbit's heart is relatively difficult to throw into fibrillation, it was decided to use this animal in the majority of the experiments, and, in the light of the results thus obtained, to repeat the observations on a sufficient number of cats and dogs to make it possible to form an opinion as to how far they might be applicable to man.

Equipment

With the excellent electrical facilities supplied by the Hydro-Electric Power Commission of Ontario (Fig. 1) we were able to obtain currents of any desired magnitude. The commercial 25-cycle 110-volt alternating current came in through 30-ampere fuses and was connected, through a time switch controlled by a potentiometer, to a 3-kilowatt transformer with a ratio of 10:1. On the high tension side, a water resistance which could be varied between 100 and 11,000 ohms was inserted. In series with the grounded side of the high tension circuit, a 3-ampere ammeter was connected. For the preliminary experiments, a 75 to 150 range alternating voltmeter with a potential transformer having a ratio of 10:1 was used. With this equipment, we were able to deliver at our electrodes a current of very definite strength for a definite time. We were, furthermore, enabled to measure the voltage and the amperage during the actual passage of the current through the animal. With reasonable care in the adjustment of the

electrodes, we could duplicate the experimental conditions very readily.

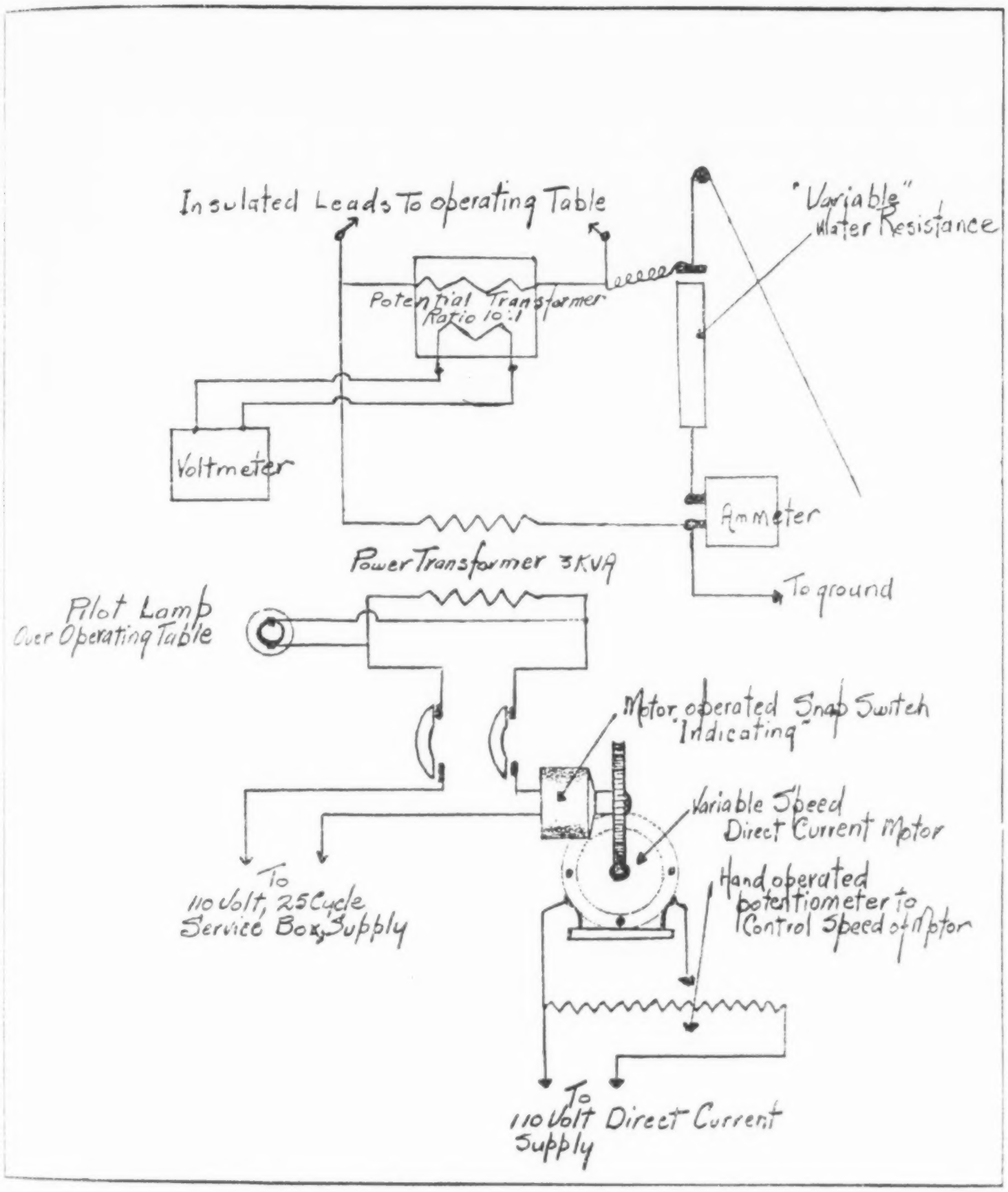
Insulated Leads To operating Table
"Variable" Water Resistance
Potential Transformer Ratio 10:1
Voltmeter
Ammeter
Power Transformer 3KVA
To ground
Pilot Lamp Over Operating Table
Motor operated Snap Switch "Indicating"
Variable Speed Direct Current Motor
Hand operated potentiometer to Control Speed of Motor
To 110 Volt, 25 Cycle Service Box Supply
To 110 Volt Direct Current Supply

FIG. 1.—Diagram illustrating electrical detail used throughout the work.

Effects of Current

The results were found to vary considerably according to the parts of the body to which the electrodes were applied and according to the exact design of the electrodes. For the first series of experiments (Table 1) and in a few preliminary experiments, the electrodes consisted of two flexible strips of copper, one being 2 by 4 cm. and the other square with an area of

2.25 sq. cm. The former was wrapped tightly around a previously shaved and moistened hind limb, while the latter was curved slightly and fitted on to the shaved occiput. According to Prevost and Battelli and other observers, a high tension current traversing the body between these electrodes will ordinarily miss the heart but will cause disturbances in the brain and the spinal cord. A total of sixty experiments with electrodes of this type were performed on thirty-three animals. The primary effects of the shocks on each animal have been tabulated in Table 1.

Analysis of this table shows that in nine of the thirty-three cases spontaneous respiration occurred following the application of the electric current. In six others, artificial respiration brought about recovery. The remaining eighteen animals died. In one-half of these the heart stopped beating within one minute after the cessation of the current, while in five of the seven to which artificial respiration was applied the heart continued to beat for from two to nine minutes. The final four of the fatal cases in which no artificial respiration was employed and in which the heart beat persisted for from two and one-half to three and one-half minutes are grouped by themselves since the probable factor causing death was uncertain. For the other fourteen fatal cases, it seems to be fairly evident that the heart, quite apart from fibrillation, had been damaged in some way by the current, since it stopped beating either within one minute of the application of the electric current or at a later interval in spite of continued artificial respiration. We would conclude, therefore, that when a current of high tension traverses the body from the head to a posterior limb, the heart is injured in approximately 45 per cent. of the cases. Of the others (55 per cent.) some die of respiratory failure, and it is probable that in those which survive very little of the electric current can have passed directly through a vital center. It is to be noted that the ratio of these figures may be somewhat related to that of Scott Ram's figures which have been quoted above.

These observations demonstrate that when the electric current traverses the length of the body, it is impossible to predict whether or not the heart will be affected. We therefore undertook a second series of experiments in which the electrodes were applied to the head in such a manner that the current would pass through the vital centers of the brain, the object being to determine the exact condition of current strength and the duration causing death when these centers alone are involved.

Using the same electrodes as in the previous series of experiments and placing them in various situations on the head and neck, we found that we were unable to bring about a permanent cessation of respiration. The current was evidently being shunted around the bony skull through the subcutaneous tissues. Electrodes of a different type were then devised and used in all the succeeding experiments.

One of the electrodes consisted of a straight piece of No. 8 copper wire about 2 inches long, which was inserted well back in the nasal cavity. The second electrode was arranged to fit snugly on the exposed atloido-occipital ligament. It consisted of a small brad

insulated to its tip and attached to the bend of a piece of heavy copper wire shaped in the form of a horseshoe. The tip of this electrode was about

TABLE 1.—PRIMARY EFFECT OF SHOCK ON THIRTY-THREE RABBITS WHEN ELECTRODES CONSISTING OF TWO FLEXIBLE STRIPS OF COPPER WERE USED,[1] ONE APPLIED TO THE HIND LIMB AND THE OTHER TO THE OCCIPUT

RABBIT NO.	STRENGTH OF CURRENT *milliamp.*	DURATION OF CURRENT *sec.*	SURVIVAL OF HEART *min./sec.*	REMARKS
9	1,200	4	Recovered	
13	1,000	2	"	
14	1,500	2	"	
20	1,600	3	"	Spontaneous onset of respiration after cessation of current without artificial respiration.
22	1,500	3	"	
23	1,600	3	"	
25	1,800	3	"	
30	1,700	3	"	
34	1,700	4	"	
15	1,700	2	"	
18	1,600	3	"	
24	1,700	3	"	Recovery after artificial respiration.
27	1,600	3	"	
28	1,600	3	"	
31	1,800	3	"	
		Deaths		
11	2,000	2	9 /—	
16	1,600	3	3 /—	
17	1,600	3	5 /—	
21	1,700	3	3 /—	Artificial respiration applied.
36	1,800	4	2 /—	
6	2,200	3	— /25	
10	1,700	2	1 /—	
7	600	2	— /30	
8	1,500	2	— /45	
12	1,800	2	— /2	
19	2,000	3	1 /—	
26	1,800	3	— /20	
29	1,800	3	1 /—	No artificial respiration applied.
38	1,700	4	— /45	
32	1,800	3	3 /30	
33	1,800	4	3 /—	
35	1,800	4	2 /25	
37	1,800	4	2 /30	

[1] The electrode applied to the hind limb was 2 cm. by 4 cm.; that applied to the occiput was square, with an area of 2.25 sq. cm.

0.1 sq. cm. in area and it was maintained in the proper position by elastic bands passing through the animal's mouth and hooked on the curved arms of the horseshoe shaped support. The tension of the elastic bands kept the the electrode firmly in place, thus insuring proper contact at all times.

For this series of experiments, it was necessary to anesthetize the animal. There has been some discussion as to the "protective action" afforded by an anesthetic. It is doubtful if it exists. We have attempted to avoid this possible complicating factor by waiting after the initial operations until the anesthesia had become less profound. Thus, we have always been careful to obtain the eye reflex before administering the electric current although the animal was obviously unconscious. We do not think that our results have been seriously influenced on account of anesthesia, since

TABLE 2.—RESULTS OF EXPERIMENTS ON TWENTY-ONE ANIMALS IN WHICH RESPIRATION DID NOT RETURN AND ARTIFICIAL RESPIRATION WAS NOT ATTEMPTED, AND WHICH SHOWED NO EVIDENCE OF GROSS INJURY TO THE BRAIN OR THE SPINAL CORD

ANIMAL NO.	STRENGTH OF CURRENT *milliamp.*	DURATION OF CURRENT *sec.*	SURVIVAL OF HEART *min./sec.*
Small Electrode[1]			
Rabbit 41	1,600	3	5/—
" 42	1,400	3½	4/—
" 43B	1,200	3	4/45
" 45	800	3	6/—
" 46B	800	3	5/45
" 48	800	3	3/—
" 50	500	2	4/45
" 54C	600	1½	6/—
" 65	600	2	4/—
" 68	600	2	4/45
" 69	700	2	5/—
" 70	600	2	5/15
Large Electrode[2]			
Rabbit 76B	1,200	2	3/—
" 77	1,100	2	6/—
" 78	1,200	2	4/30
" 79	1,200	2	3/30
" 80	1,200	2	5/—
" 93	600	2	4/—
Cat 1B	1,200	2	4/30
" 2B	1,600	2	6/—
" 7	1,700	2	7/—

[1] Area, 0.1 sq. cm.
[2] Area, 0.36 sq. cm.

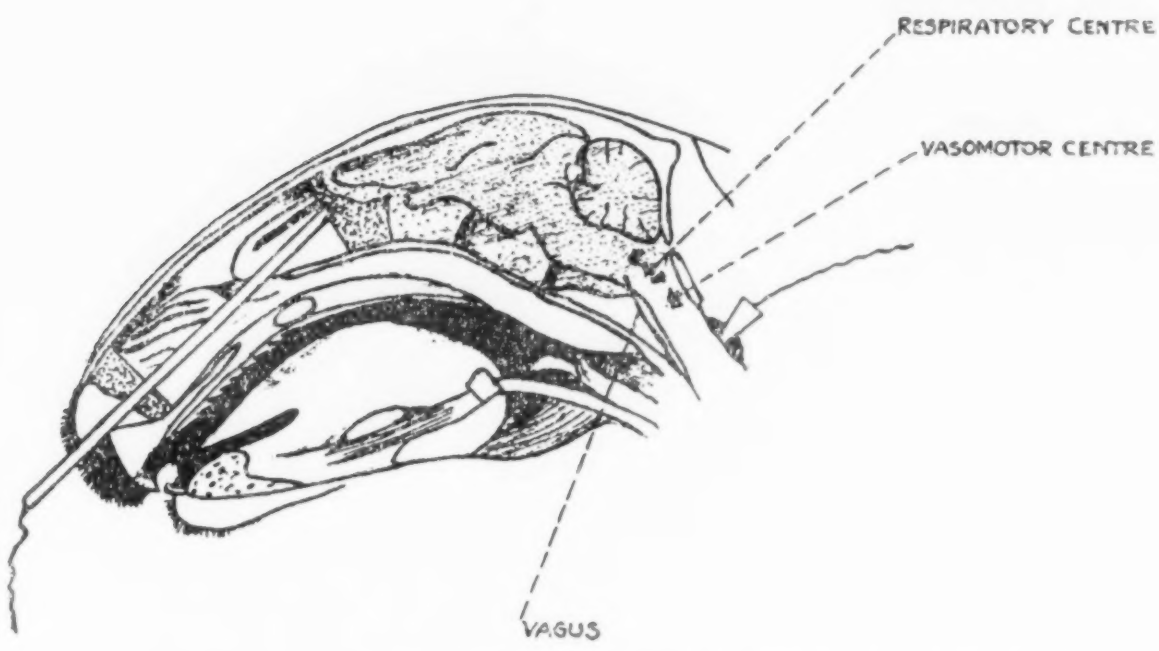

FIG. 2.—A vertical section through a rabbit's head, showing the location of the electrodes and the approximate location of the chief centers involved in the path of the current.

TABLE 3.—RESULTS OF EXPERIMENTS ON EIGHTEEN ANIMALS IN WHICH RESPIRATION RETURNED AND NO ARTIFICIAL RESPIRATION WAS ATTEMPTED, AND WHICH SHOWED NO EVIDENCE OF PITHING

ANIMAL NO.	STRENGTH OF CURRENT *milliamp.*	DURATION OF CURRENT *sec.*	RETURN OF VOLUNTARY RESPIRATION *min./sec.*
	Small Electrode[1]		
Rabbit 43	1,200	3	—/30
" 46	600	3	—/3
" 49	300	3	—/30
" 54	400	2	Immediately.
" 54B	500	2	Immediately.
" 55	300	2	—/2
" 61	500	2	1/10
" 64	300	2	—/20
" 66	600	2	—/10
" 66B	800	2	—/10
	Large Electrode[2]		
Rabbit 72	1,000	2	1/5
" 72B	1,100	2	—/30
" 75B	1,200	2	—/1
" 75C	800	2	1/45
" 94	700	2	Immediately.
" 96	600	2	1/45
Cat 2	1,200	2	Immediately.
" 8	1,500	2	1/—

[1] Area, 0.1 sq. cm.
[2] Area, 0.36 sq. cm.

there has been no evidence that the depth of this affects them.

TABLE 4.—RESULTS OF EXPERIMENTS IN WHICH THE CURRENT WAS APPLIED FOR TWO SECONDS

RABBIT NO.	STRENGTH OF CURRENT	RETURN OF VOLUNTARY RESPIRATION		DEATH AS JUDGED BY CESSATION OF HEART[1]
		Without Application of Artificial Respiration	With Application of Artificial Respiration	
	milliamp.	*min./sec.*	*min./sec.*	*min./sec.*
		Small Electrode[2]		
64	300	—/20	....	....
55	300	—/2	....	....
51	400	....	9/30	....
54	400	—/2	....	....
54B	500	—/2	....	....
61	500	1/10	....	....
50	500	....	....	4/45
49	500	....	6/—	....
55B	500	....	8/—	....
56	500	....	8/—	....
58	500	....	....	Pithed.
66C	600	—/10	....	....
54C	600	....	....	6/—
65	600	....	....	4/—
68	600	....	....	4/45
70	600	....	....	5/15
61B	600	....	14/—	....
69	700	....	....	5/—
62	700	....	3/20	....
66B	800	—/10	....	....
62B	800	....	5/—	....
63	800	....	8/—	....
63B	800	....	....	Pithed.
67	800	....	9/—	....
66	900	....	4/—	....

[1] No artificial respiration applied.

[2] Area, 0.1 sq. cm.

The procedure was as follows: The animal was anesthetized and cannulas were inserted in the trachea and into the carotid artery. The atloido-occipital ligament was then exposed, the special electrode placed in position, and the other electrode inserted in the nasal cavity. After allowing the animal to recover somewhat from the anesthesia, the desired electric current was passed between the electrodes for a definite time. Figure 2 illustrates in a diagrammatic way the path of the current and the location of the vital centers which it affects.

In Table 2 we have tabulated the results of experiments on eighteen rabbits in which the heart beat ceased

within a few minutes after receiving electric currents which varied in strength from 500 to 1,600 milliamperes and were applied for one and one-half to three and one-half seconds. On the application of the current, the animals went into a generalized contraction which was maintained for the duration of the shock. When the current was broken, a few clonic convulsions occurred, followed in a few seconds by complete muscular relaxation during which the mucous membranes became dark and the pupils dilated. At a later interval (in from three to six minutes) the heart could no longer be heard. Subsequent exposure of the heart revealed no evidence of fibrillation and at the point of contact of the electrodes there was no visible destruction of tissue. Apparently death in these cases was purely of the asphyxial type.

In a certain number of instances, the electric current did not cause death, and respiration returned voluntarily

TABLE 4.—*Concluded*

RABBIT NO.	STRENGTH OF CURRENT	RETURN OF VOLUNTARY RESPIRATION		DEATH AS JUDGED BY CESSATION OF HEART[1]
		Without Application of Artificial Respiration	With Application of Artificial Respiration	
	milliamp.	*min./sec.*	*min./sec.*	*min./sec.*
		Large Electrode[3]		
93	600	....	....	4/—
96	600	1/45	....	....
96B	600	....	2/15	....
94	700	Immediately	....	....
75C	800	1/45	....	....
92	800	....	6/15	....
73	820	....	5/—	....
72	1,000	1/5	....	....
72C	1,000	....	....	Pithed.
72B	1,100	—/30	....	....
77	1,100	....	....	6/—
75B	1,200	—/1	....	....
76B	1,200	....	....	3/—
78	1,200	....	....	4/30
79	1,200	....	....	3/30
80	1,200	....	....	5/—
76	1,200	....	3/20	....
81	1,200	....	3/45	....
82	1,200	....	7/—	....
74	1,500	....	....	Pithed.
81B	2,000	....	....	Pithed.[4]
84	2,000	....	....	Pithed.[4]
83	2,300	....	....	Pithed.[4]

[3] Area, 0.36 sq. cm.
[4] Artificial respiration applied.

after a variable time had elapsed. These have been listed in Table 3. Comparing this with Table 2, it may be seen that in the majority of instances the fatal cases received more current than did the nonfatal. In the case of amperages which might or might not be fatal, it is possible that the electrodes were so placed that the mass of the electric current missed the vital centers. There does appear, however, to be a more or less definite border line between fatal and nonfatal amperages and one can say that with the smaller electrode the fatal amperage is between 600 and 700 milliamperes applied for two seconds. This is perhaps better illustrated in the experiments shown in Table 4 in which the time of application of the current and the method of applying the electrodes were kept constant and the results have been grouped according to the number of amperes which were passed through the animal. The figures show that the proportion of these which died or presumably would have died, had artificial respiration not been applied, increases approximately parallel with the amperage until a point is reached where gross damage to the tissue takes place. This damage, as will be shown later, always occurs when the capacity of the contact area is not sufficiently large to carry the current. When there is no damage to the cord, it may be said that the seriousness of the result is proportional to the amount of current which the animal receives through its vital centers—*viz.*, respiratory, vasomotor, etc.

In this connection it may be noted (Tables 2 and 4) that fatal apnea was produced by electric currents varying greatly in amperage when the size of the electrodes on the atloido-occipital membrane was also varied. For example, when the electrode with an area of 0.1 sq. cm. was used, a current of 600 milliamperes was sufficient to cause a fatal apnea. When the area was increased to 0.36 sq. cm., 1,200 milliamperes were required. The explanation no doubt lies in the fact that, in any conductor, the current density at any point is directly proportional to the area at that point. Therefore, the smaller the electrode, the more concentrated is the current and the greater is the damage to the tissues in its path.

Artificial Respiration

Having determined the approximate conditions under which fatal results are to be expected, we then proceeded to study the effect of artificial respiration on animals that had been given a shock which, in the vast majority of instances, was of sufficient amperage to be fatal. A total of thirty such experiments are collected in Tables 5 and 6.

Artificial respiration was applied in from one to five minutes after the electric current had been switched off and was maintained until either recovery had taken place or definite evidence of death was established. We were able to bring about recovery in twenty-five cases (Table 5) and failed to do so in five (Table 6). In those which recovered, the first attempt at voluntary respiratory effort was observed to occur within two minutes after the shock in one-third of the cases, while in the remainder a longer interval elapsed—as much as twelve minutes in one case. The respirations were at first irregular and

gasping in character but in a few minutes they gradually became more regular and approximated the normal. In some instances, notably those in which the initial voluntary respirations were the most delayed, the gasps succeeded one another at the same rate as that at which the artificial respiration had been applied. When sufficient recovery had taken place and artificial

TABLE 5.—RESULTS OF EXPERIMENTS ON TWENTY-FIVE ANIMALS IN WHICH RESPIRATION RETURNED AFTER ARTIFICIAL RESPIRATION WAS APPLIED, AND WHICH SHOWED NO EVIDENCE OF PITHING

ANIMAL NO.	STRENGTH OF CURRENT	DURATION OF CURRENT	TIME ARTIFICIAL RESPIRATION WAS BEGUN AFTER CESSATION OF CURRENT	RETURN OF VOLUNTARY RESPIRATION AFTER APPLICATION OF ARTIFICIAL RESPIRATION	TOTAL TIME FROM CESSATION OF CURRENT
	milliamp.	*sec.*	*min./sec.*	*min./sec.*	*min./sec.*
Small Electrode[1]					
Rabbit 49B	500	1½	4 /—	2 /—	6 /—
" 51	400	2	5 /—	4 /30	9 /30
" 55B	500	2	4 /—	4 /—	8 /—
" 56	500	2	4 /—	4 /—	8 /—
" 61B	600	2	2 /—	12 /—	14 /—
" 62	700	2	2 /20	1 /—	3 /20
" 62B	800	2	2 /—	3 /—	5 /—
" 63	800	2	2 /—	6 /—	8 /—
" 66C	900	2	2 /—	2 /—	4 /—
" 67	800	2	3 /—	6 /—	9 /—
Cat 4	900	2	2 /—	1 /20	3 /20
Large Electrode[2]					
Rabbit 73	820	2	2 /—	3 /—	5 /—
" 75	600	Momentary	3 /—	2 /30	5 /30
" 76	1,200	2	1 /50	1 /30	3 /20
" 81	1,200	2	3 /—	— /45	3 /45
" 82	1,200	2	2 /—	5 /—	7 /—
" 85	1,200	3	Immediately	9 /—	9 /—
" 86	1,500	3	1 /—	— /30	1 /30
" 89	600	4	2 /15	— /15	2 /30
" 90	600	5	2 /—	1 /—	3 /—
" 92	800	2	2 /15	4 /—	6 /15
" 96B	600	2	1 /45	— /30	2 /15
Cat 1	1,200	2	1 /40	1 /20	3 /—
" 3	1,700	2	1 /45	2 /—	3 /45
" 6	1,100	2	3 /45[3]	4 /45	8 /30

[1] Area, 0.1 sq. cm.
[2] Area, 0.36 sq. cm.
[3] Artificial respiration was accompanied by heart massage.

respiration could be withdrawn, the respirations gradually became normal

ligament had been badly charred and in some cases had been burned. The

TABLE 6.—RESULTS OF EXPERIMENTS ON TEN ANIMALS IN WHICH RESPIRATION DID NOT RETURN, AND WHICH SHOWED GROSS LESIONS AT AUTOPSY

ANIMAL NO.	STRENGTH OF CURRENT *milliamp.*	DURATION OF CURRENT *sec.*	TIME ARTIFICIAL RESPIRATION WAS BEGUN AFTER CESSATION OF CURRENT[1]	GROSS LESION AT AUTOPSY
Small Electrode[2]				
Rabbit 58	500	2	Not applied	Cord burned.
" 63B	800	2	Not applied	Cord burned.
" 64B	700	2	1 min.	Cord burned.
Large Electrode[3]				
Rabbit 72C	1,000	2	Not applied	Pithed.
" 74	1,500	2	Not applied	Pithed.
" 81B	2,000	2	2 min.	Severe burn and hemorrhage.
" 83	2,300	2	Immediately	Slight burn on cord; pithed.
" 84	2,000	2	Immediately	Pithed.
" 88	500	3	Immediately	Pithed.
Cat 4B	1,000	2	Not applied	Pithed.

[1] In the cases where artificial respiration was not applied, it was obvious that pithing had taken place.

[2] Area, 0.1 sq. cm.

[3] Area, 0.36 sq. cm.

in character. It is evident that in the delayed cases the depression of the respiratory center was more pronounced than in the others in which the period of apnea was shorter. The significant thing is, however, that artificial respiration adequately applied was sufficient to bring about recovery in about 75 per cent. of the cases.

In the five cases in which artificial respiration failed (Table 6), subsequent examination revealed that gross damage had been done to the spinal cord or the medulla. The atloido-occipital head of the electrode had in some instances penetrated half through the cord. This damage we shall describe as pithing, and in fact the animal in such cases behaved in all respects like a so-called "pithed" animal.

Pithing occurs, as we have stated above, whenever the capacity of the contact area is insufficient to carry the current, or, in other words, when the resistance at this contact point does not allow the current to flow freely. This contact area is composed on the one side of the atloido-occipital membrane, the resistance of which will

become increased as a result of drying, such as may occur after prolonged application of the current. One would therefore expect that with the larger electrode (0.36 sq. cm. in area) a greater volume of current could be carried without causing damage because of the decreased concentration of current per unit area. On the other hand, the passage of an electric current through a conductor of relatively high resistance (such as tissue) generates heat and as a result charring occurs, and the greater the resistance the more quickly does this occur. Thus a current of 1,000 milliamperes flowing for two seconds through an electrode having an area of 0.1 sq. cm. frequently produces pithing, while with an electrode with an area of 0.36 sq. cm. it requires at least 2,000 milliamperes, applied for the same length of time, to produce a similar result.

The limitations introduced by this factor became of importance in our next experiments, which were planned for the purpose of studying the effect of remedial measures other than artificial respiration. Since it was necessary to apply an electric current which would so injure the respiratory center that artificial respiration in itself was ineffective in restoring the animal, the current was increased, but usually when 2,000 to 2,500 milliamperes were given pithing occurred even when the larger electrode (0.36 sq. cm. in area) was used. Pithing also occurred when a current of only 500 to 600 milliamperes was applied for a longer period (three to five seconds). We were, therefore, forced to abandon this method of procedure.

We were satisfied, however, that the current which we were able to employ without causing gross damage to the spinal cord was insufficient to injure permanently the respiratory center and that prompt and efficient application of artificial respiration practically always assured recovery of the animal, provided it was started within five or six minutes following the application of the shock, since at longer intervals irremediable damage had been done to the heart. It was further found necessary to maintain the artificial respiration for a considerable period of time after the first appearance of breathing by the animal itself. In some instances in which the application of artificial respiration had resulted in a return of natural breathing, it was noted, for example, that immediate withdrawal of the artificial measures was followed by the cessation of the animal's own efforts (Chart 4). Reapplication of artificial respiration in such cases was usually successful in bringing about complete recovery. In other cases where the current was not so great, the momentary application of artificial respiration was all that was necessary to restore natural breathing. One cannot emphasize too much the necessity of early and continued application of artificial respiration in electric accidents.

Counter Shock

There has been much discussion in the past with regard to the efficacy of so-called "counter shock" or "counter stimulation." The men in the field have repeatedly observed that the victim who falls from a height, following an electric shock, is more readily resuscitated than one who does not have such a fall. This has been attributed to the mechanical stimula-

tion resulting from the body's striking the ground. This idea has led to the employment of some rather drastic measures of treatment. The evidence of the value of such treatment has never been convincing and the recent work of Campbell and Leonard Hill (8) goes far to discredit it. We have further evidence which confirms this view.

Since counter shock by mechanical means must act by bringing about stimulation of sensory nerves with a resulting reflex excitation of the respiratory and other vital centers, our experiments have consisted in stimulation, with faradic shocks of moderate intensity, of the central end of the sciatic nerve in rabbits which had received apparently fatal electric shocks in the manner described above. No recoveries were obtained when artificial respiration was not at the same time applied. Furthermore, no evidence could be obtained, either in the behavior of the animal itself or in the arterial blood pressure tracings, to show that such stimulation was having any reflex effects (Chart 6). Even in those cases in which the animal was kept alive by artificial respiration it was impossible to detect any reflex effects on blood pressure or any improvement in the rate of recovery. We were, therefore, forced to conclude that counter stimulation has no demonstrable effect on resuscitation from electrocution in laboratory animals.

Graphic Records

Graphic records of the carotid blood pressure and of the respiratory movements were taken in most of the experiments throughout this work. The curve of the blood pressure during and following application of the current may be divided into several phases. These are shown in Chart 1, in which the time is also indicated in one-second intervals. The abscissa is indicated on each chart. We shall discuss first the general behavior of the tracing in each group of experiments and then consider the causal factor for each of the phases.

Chart 1 (that of rabbit 61) is typical of the group in which spontaneous respiration took place following the passage of the current. After applying the smaller electrode (0.1 sq. cm.) to the exposed atloido-occipital membrane, 500 milliamperes were given for two seconds. During that time (phase 1) the animal was in a general tonic convulsion and the heart was stopped. When the current was broken, complete muscular relaxation followed and the blood pressure fell momentarily (phase 2) and then rose rapidly to about twice its normal height (phase 3). During the rise two or three irregular beats of the heart can be made out in the tracing, but at the peak they suddenly change to a rapid, regular rhythm. The blood pressure soon fell again until it stood at about one-half the normal level (phase 4), at which it remained for about five seconds, when a slight rise again occurred (probably due to asphyxia). After about one and a quarter to two minutes, the deep indentations which mark the return of gasping respirations became evident on the curve (phase 5), and as these became more regular the blood pressure gradually returned to about its normal level.

Chart 2 (rabbit 77) is typical of those experiments in which a fatal

amount of current was used without any attempt at resuscitation. The animal received 1,100 milliamperes for two seconds through the larger electrode (area 0.36 sq. cm.) applied to the membrane. The behavior of the curve is much the same as in Chart 1, except that there is no evidence of the secondary (asphyxial) rise in blood pressure (phase 5). This rise is, in fact, rarely seen in this group. The cardiac pulsations gradually become very faint and cease to register on the tracing in about four minutes, although they can usually be heard with the stethoscope for about six minutes.

The effect of the application of artificial respiration on the blood pressure of animals which have received an apparently fatal shock is well shown in Charts 3 to 6. In the majority of these, it will be noted that the immediate response to the shock (*i.e.*, in phases 1, 2, and 3) is similar to that shown in Charts 1 and 2. The tracing in Chart 3 is fairly typical. In two minutes and twenty seconds, when the blood pressure had fallen to within a few millimeters of the base line, artificial respiration was applied. The blood pressure immediately commenced to rise and in less than one minute natural respirations set in, followed by a return of the blood pressure to or above its normal level. The other tracings vary only in the rate of response to this treatment; this would appear to depend partly on the severity of the shock and partly on the promptness with which artificial respiration is commenced. Chart 4 is particularly interesting, since it shows a series of immediate responses to the repeated application for short periods of artificial respiration, each period of recovery being followed by a fall in blood pressure, until after several such trials natural breathing set in and the pressure was sustained. It would appear that the primary effect of artificial respiration is to supply to the muscles of the heart properly oxygenated blood and thus provide for its improved action. An equally important effect is, of course, the concurrent supply of oxygenated blood to the centers of the brain, assisting materially in relieving the block to those centers.

Cause of Phenomena Observed

Experiments were now undertaken to determine, if possible, the exact cause of these phenomena. With this object in view, while the blood pressure tracings were being taken the heart was directly observed through a window in the chest wall. Rabbits, cats, and dogs were used in these observations with essentially similar results.

Circulatory System.—During the period of the passage of the electric current (phase 1), the heart was seen to be completely inhibited in the rabbit and the dog, while in the cat the inhibition was not so complete although the beats were not of sufficient force to register on the tracings. This inhibition of the heart is the result of stimulation of the vagus centers, as was demonstrated by the following experiments. A rabbit was prepared in the usual manner and the vagi were dissected and severed at the level of the cricoid cartilage. On the passage of the current, the heart was found to be inhibited to a much less degree than when the vagi are intact. Sufficient

atropine was then injected intravenously to block effectively the vagus nerve endings in the heart (as tested by stimulation of the peripheral end of the vagus). In a few minutes after the injection, the heart was in no wise affected by reapplication of current through the electrodes. This would show that when the vagi are intact the heart is inhibited through stimulation of the vagus center. The fact that following section of the vagi some degree of inhibition is obtained is evidence that there are extravagal inhibitory fibers to the heart. When these are controlled by the administration of atropine no inhibition is obtained on the passage of the current.

The passage of the current also paralyzes the vagus center itself so that the heart cannot be inhibited reflexly for some time after its passage. In the tracings of Chart 5 it can be seen that, before any current was applied, stimulation of the central end of one vagus nerve caused a distinct (reflex) fall in blood pressure. After application of the current, natural breathing was absent and artificial respiration had to be applied. During this stage, stimulation of the vagus, as before, had no effect on the blood pressure, nor did any effect become evident until some time after normal breathing had returned and the conjunctival reflex could be elicited. This result is interpreted as showing that a complete block had been established at the vagus nerve center, as a result of the passage of the current.

It was usually observed that, in addition to a more or less complete inhibition of the heart during the passage of the current, the venous side of the circulation becomes greatly distended with blood, this condition resulting from the combined effects of the generalized tonic contraction of all the muscles in the body and stimulation of the vasoconstrictor centers in forcing the blood into the venous side of the circulation. The sudden cessation of this tonic spasm, when the current is broken, momentarily reduces the pressure on the arterial side so that the blood pressure falls (phase 2).

The rapid increase in blood pressure which immediately follows this momentary fall (phase 3) occurs because the blood which had accumulated in the venous side is being forced into the arterial side of the circulation, and while this is happening the heart beats are very powerful. That the whole arterial tree is affected is shown by an increase in kidney volume synchronous with the rise in blood pressure. As soon as the excess of load has been delivered, the heart beats become relatively normal in character but the rate is greatly increased. It may be interesting to note here that the increase in blood pressure is never so marked in the cat as it is in the rabbit and the dog. This is probably due to the fact that the cat's heart is not completely inhibited during the passage of the current. It may be noted, also, that this rapid increase in blood pressure is a probable cause of many of the capillary hemorrhages which microscopic examination reveals in the brain and the spinal cord.

The blood pressure remains at its high level for a few seconds and then falls more or less rapidly until the basal level is reached (phase 4). The fall in pressure which soon follows the rise (phase 4) is due, primarily, to a generalized vasodilatation resulting

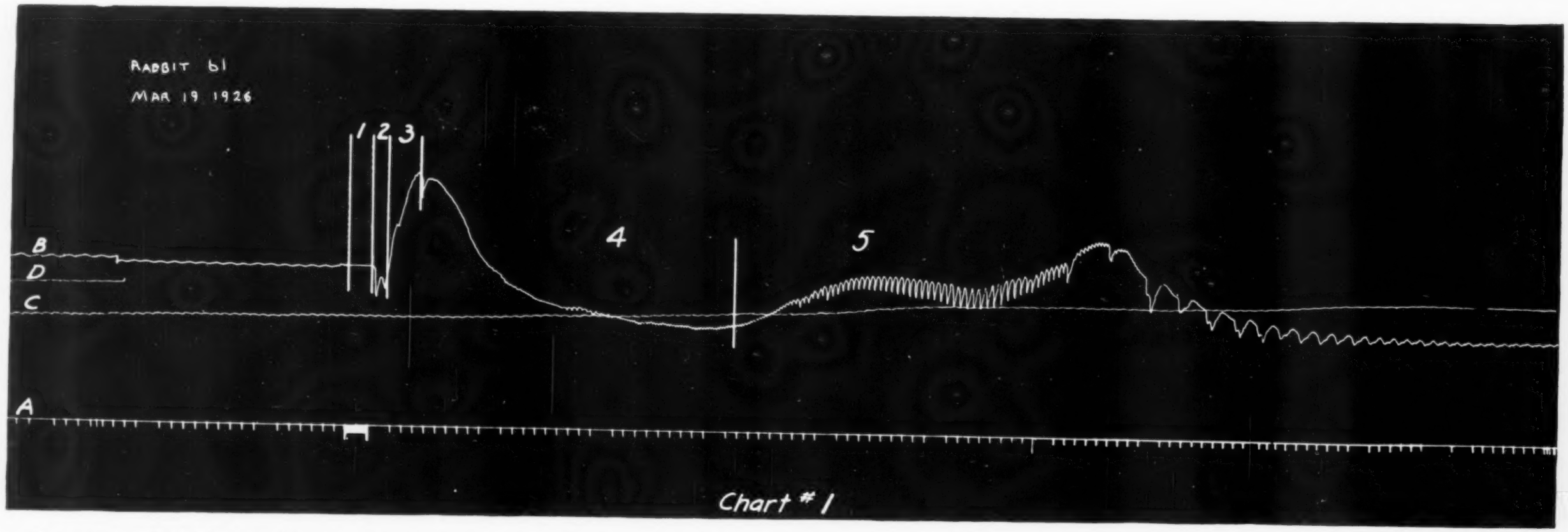

CHART 1.—Rabbit 61. Showing recove y of animal without artificial respiration. Electrodes on the atloido-occipital membrane and in the nose. Current 500 milliamperes for two seconds.

A = time and blood pressure base line. B, C, and D = first, second, and third lines of tracing. The tracings are taken on a circular drum so that line B is continuous with line C, and so on.

The parallel vertical lines indicate the phases into which the tracings have been divided as described in the context. The blocked-out portion of the time and blood pressure base line indicates the time of the passage of the electric current.

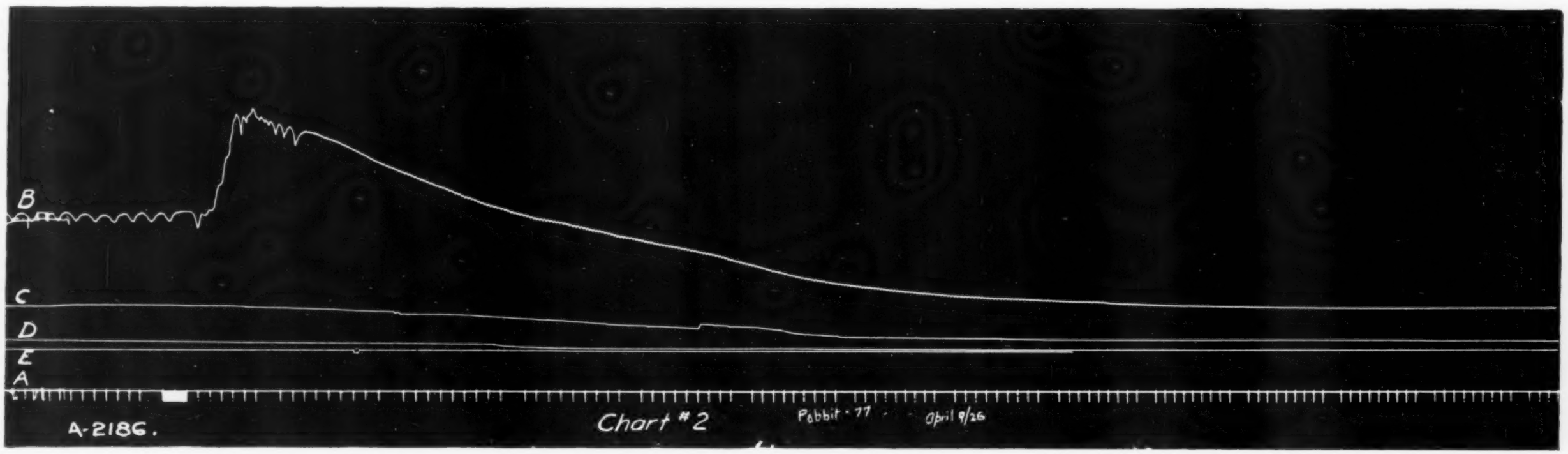

CHART 2.—Rabbit 77. Showing death of animal when artificial respiration is not applied. Electrodes on the atloido-occipital membrane and in the nose. Current 1,100 milliamperes for two seconds.
A = time and blood pressure base line. B, C, D, and E = first, second, third, and fourth lines of tracing.

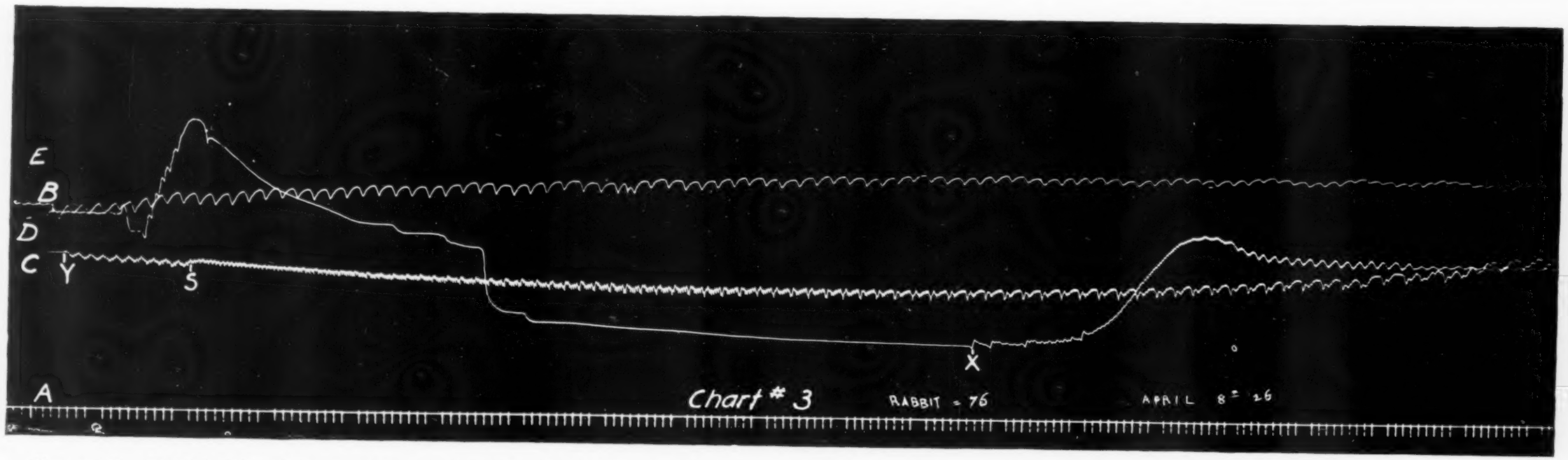

CHART 3.—Rabbit 76. Return of normal respiration following the application of artificial respiration in an animal receiving a lethal dose. Electrodes on the atloido-occipital membrane and in the nose. Current 1,200 milliamperes for two seconds.

A, B, C, D, and E as in previous charts. X = application of artificial respiration. Y = return of the animal's own respiration. S = artificial respiration discontinued.

Note.—The usual rapid fall in blood pressure (phase 4) in this chart is distorted by a momentary twist in the carotid cannulas.

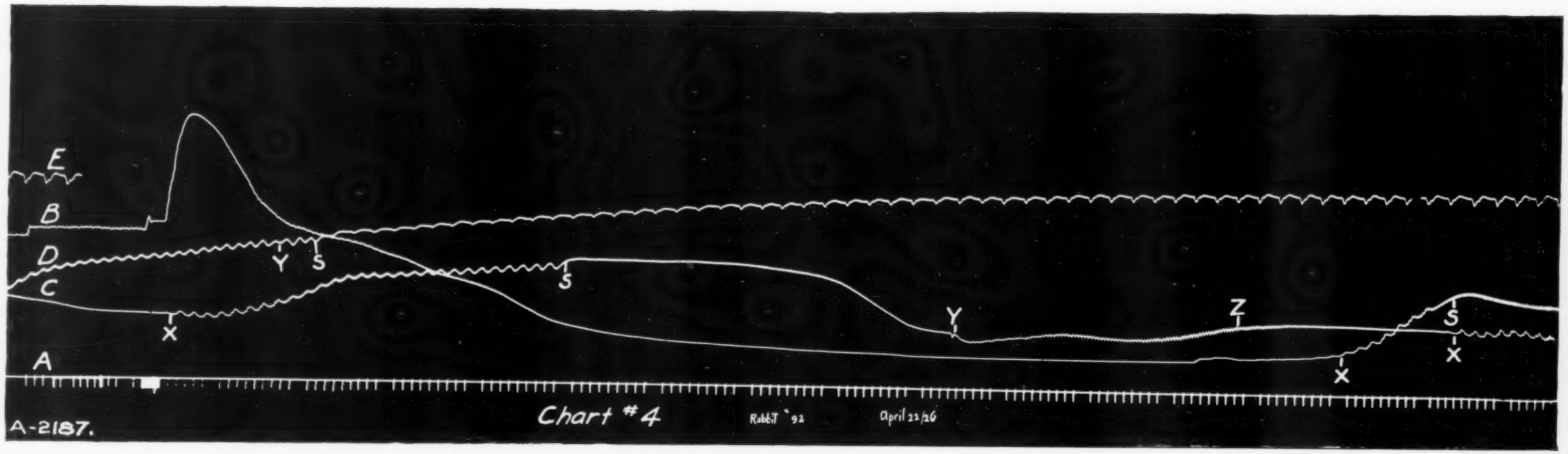

CHART 4.—Rabbit 92. The effect on resuscitation of interruptions in the application of artificial respiration in an animal showing ultimate recovery. Electrodes on the atloido-occipital membrane and in the nose. Current 800 milliamperes for two seconds.
A, B, C, D, and E as in previous charts. X, Y, and S as in Chart 3. Z = failure of animal's own respiration.

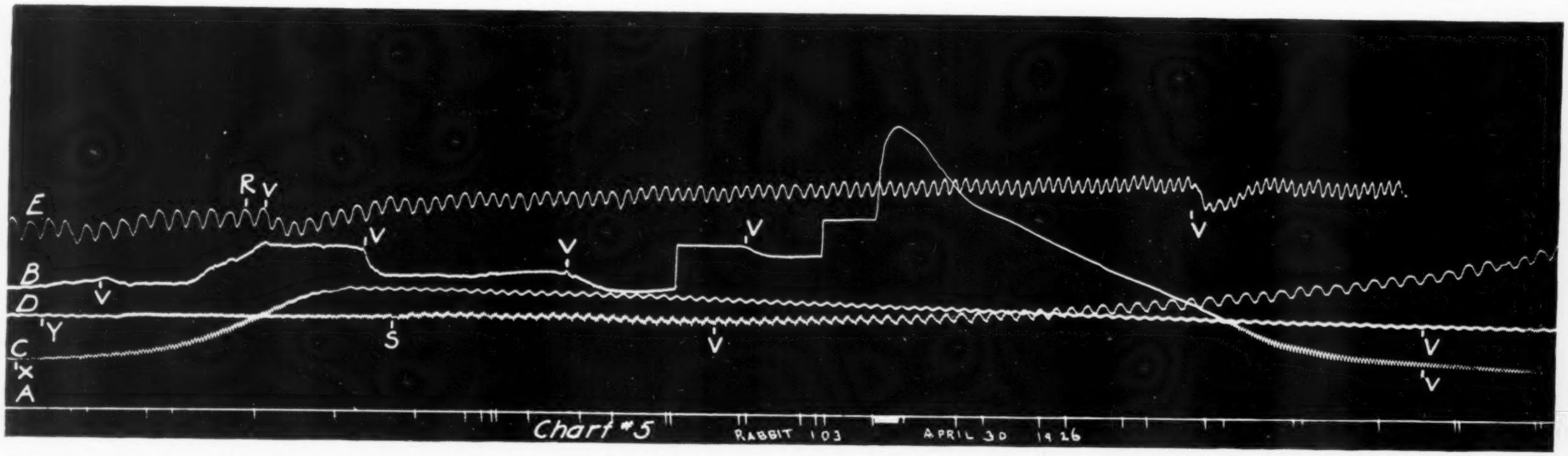

Chart 5.—Rabbit 103. Demonstrating the presence of a block in the centers following the application of an electric current. Electrodes on the atloido-occipital membrane and in the nose. Current 700 milliamperes for two seconds.

A, B, C, D, and E as in previous charts. X, Y, Z, and S as in previous charts. V = stimulation of the central end of the cut vagus. R = return of eye reflex.

Note.—The reflex response to stimulation of the central end of the cut vagus is absent following the passage of the current. After a period of approximately eight minutes, during which artificial respiration was applied and the respiratory center became active, this reflex returns, and for the first time an eye reflex can be obtained.

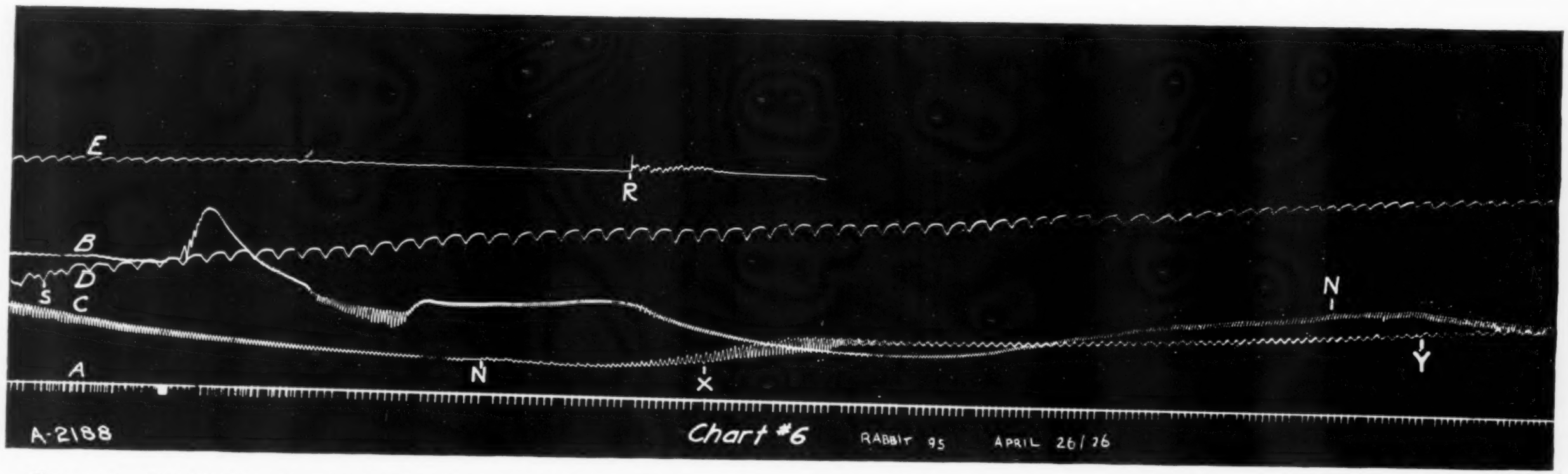

CHART 6.—Rabbit 95. Demonstrating that stimulation of afferent nerves has no influence on the recovery of the animal following electric shock. Electrodes on the atloido-occipital membrane and in the nose. Current 600 milliamperes for two seconds.
A, B, C, D, and E as in previous charts. X, Y, S, and R as in previous charts. N = stimulation of the central end of the cut sciatic nerve.

from inhibition of the vasoconstrictor nerve center similar to the inhibition described in the respiratory center. The result of this can readily be demonstrated by kidney volume tracings and by direct observation of the vessels, which are usually found to be markedly dilated at this time. We consider this inhibition of the vascular tone to be of the greatest importance as a cause of death, since the blood thereby stagnates in the peripheral blood vessels. The systemic circulation becomes embarrassed because of the inadequate blood supply to the heart and the heart beats with rapidly decreasing force till in five or six minutes it stops entirely.

In an animal which has received a sublethal dose of current this paralysis of vasomotor tone is not complete, and frequently a secondary rise in blood pressure accompanies the onset of asphyxia (phase 5). In such cases voluntary respiratory efforts usually appear shortly after the recovery in blood pressure, although in some cases artificial respiration may have to be applied. The most probable explanation of this rise is that the vasoconstrictor and the respiratory centers have not been so profoundly affected but that they can be stimulated by the accumulation of carbon dioxide in the blood.

Nervous System.—In an endeavor to determine whether any constant pathologic lesion of the nervous system is associated with the passage of the electric current in the manner described, the brains of the majority of the animals were removed and sectioned in paraffin by D. J. Bowie. Since the phenomena produced by the current are apparently those of a more or less temporary nerve block, it was not expected that any typical lesion would be found. It has been demonstrated that short periods of asphyxia alone produce definite changes in the structure of both sensory and motor nerve cells, those of the cerebellum and the cerebrum being the most sensitive. One must, therefore, take great care in the interpretation of similar changes before attributing them to the action of the electric current as such. For the present, we have concentrated on the occurrence of capillary hemorrhages in the brain and the spinal cord, since many writers state that this is the most constant feature of death by electric shock.

It was at first thought that these hemorrhages might possibly be associated with failure of the cardiac and the respiratory centers of the medulla, but the study of a great many sections failed to produce sufficient evidence for assuming that there is any such association. While small hemorrhages were frequently found in the proximity of the dorsal motor nucleus of the vagus nerve, they were just as frequently found in other parts of the brain and the spinal cord. Hemorrhages were somewhat more frequently found at the root of the acoustic and the facial nerves. No definite relationship could be made out, however, between the severity of the shock and the presence of these hemorrhages in any part of the brains. Animals which had received a sublethal dose of current sometimes showed on subsequent examination as severe hemorrhages as a definitely fatal case, while in some of the fatal cases no hemorrhages could be found. On the other hand, somewhat similar hemorrhages were found

in the brains of animals which had not been subjected to an electric current. When hemorrhages do occur in animals subjected to an electric current they are, therefore, as stated above, most probably due to the sudden great increase in blood pressure which follows the passage of the electric current through the body, and not to the local effect of the current. Their presence is probably not an important factor in death from electricity.

Summary

The initial effect of the passage of the alternating current through the brain is intense stimulation of all the centers in its path. There is a generalized muscular contraction which, together with a pronounced vasoconstriction, results in a great accumulation of blood in the venous side of the circulation. The stimulation of the vagus center causes inhibition of the heart, an effect which, to a large extent, can be obviated by section of both vagi. That there is not total absence of inhibition of the heart with this procedure is evidence that there are extravagal inhibitory fibers, and when these are controlled with atropine there is no inhibition of the heart on the passage of the current.

When the current is broken, this period of stimulation is followed by a period of profound inhibition or paralysis of all these centers. The animal becomes limp and unconscious; respiratory movements are absent; the eye reflex cannot be elicited; the blood pressure rises to almost twice the normal and then falls with the dilatation of the blood vessels even although the heart beats at an increased rate; there is no reflex response to sensory stimulation (stimulation of the central end of the cut sciatic nerve, Chart 6) and severe stimulation of the central end of the cut vagus has no reflex effect on the heart (Chart 5).

That this paralysis may be only temporary is shown in our experiments by the fact that artificial respiration brought about recovery in all cases in which no gross damage to the brain or the spinal cord had been done by the heat generated at the point of application of the electrodes. That gradual recovery may follow the period of paralysis is clearly shown in Charts 3 to 6. With the return of consciousness all the reflexes return, and in the majority of instances there is an interval of only a few seconds between the initial breath and their return (Charts 5 and 6). The failure to recognize a constant definite pathologic lesion in the brain or the spinal cord of an animal subjected to an electric shock is also evidence that the paralysis may be of only a temporary nature.

It seems, therefore, safe to conclude that it is the nerve centers themselves that are paralyzed by the passage of the current, so that impulses reaching them are completely blocked and the normal rhythmic discharge of the respiratory center ceases. This explains the failure of counter stimulation as an aid to resuscitation. It also explains the failure to obtain reflex vagal inhibition of the heart in the shocked animal. It is only after the application of artificial respiration with efficient aeration that the inhibition can be observed to pass off. Furthermore (as shown in Chart 4), it is exceedingly important that artifi-

cial respiration be commenced early and be maintained for a sufficient length of time.

Conclusions

1. The experiments herein described confirm the deductions of previous observers as to the cause of death in electric shock: namely, that it may be due to primary cardiac failure, to primary respiratory failure, or to a combination of both.

2. In laboratory animals, when the current traverses the body from the head to a hind limb, about 45 per cent. of the deaths are of purely cardiac origin. The remainder of the deaths occur because of failure of the respiratory movements.

3. In these, as in the group in which the current is passed directly through the brain, a condition of profound paralysis or block becomes established in the respiratory, vagus, and vasomotor centers.

4. That the block involves these centers is shown by experiments which demonstrate clearly the absence in electrocuted animals of reflex effects normally functioning through them. Since the nerve centers become insensitive to extraneous influences, counter stimulation is not an aid to recovery.

5. No definite histologic changes can be made out in the brain to account for the symptoms. The capillary hemorrhages which occur do not appear to be significant.

6. The foregoing experiments also show definitely that when the electric current does not cause charring of nerve structures the paralysis or block is recovered from and the reflexes return, provided efficient artificial respiration is applied.

7. It is of the greatest importance that artificial respiration be applied early and be maintained for a sufficient length of time. Owing to the nature of the block in the nerve centers the ordinary tests for death should not be accepted and "nothing less than cooling of the body or the onset of rigor mortis should be considered to be evidence of death."

The thanks of the writer are due to Prof. J. J. R. Macleod for his stimulating interest and helpful criticism; to Mr. Wills Maclachlan, Consulting Engineer, for his kindly advice and assistance throughout the progress of the work; to Dr. R. E. Gaby, Surgeon, for his assistance in the earlier stages of the experimental work and for notes from his clinical cases; to Mr. F. L. Harrison, Electrical Assistant, High Tension Laboratory, Hydro-Electric Power Commission of Ontario, for his assistance in the handling of the electrical facilities and for his co-operation in surmounting the numerous technical difficulties of the research; to D. J. Bowie, B.Sc., M.A., for the histologic sections; and to the numerous members of the staff of the Hydro-Electric Power Commission who were of great assistance in many ways.

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BOOK REVIEWS

PRINCIPLES OF MEDICAL TREATMENT. By *George Cheever Shattuck*, M.D., A.M., Assistant Professor of Tropical Medicine, Harvard Medical School; Junior Visiting Physician, Boston City Hospital, in Charge of the Service for Tropical Diseases; Formerly Assistant Visiting Physician, Massachusetts General Hospital; and Assistant in Clinical Medicine, Harvard Medical School. Sixth edition, revised and enlarged. With contributions by various authors. Cloth. Pp. 256 with index. Cambridge: Harvard University Press, 1926.

The author states that "the book represents an attempt to set forth clearly and concisely sound principles of medical practice based, so far as possible, upon scientific medical knowledge. Special effort has been made to emphasize principles and to describe separately the methods recommended for putting them into practice." Salient facts have also been outlined of the mode of action of diseases and disease groups in order to correlate treatment. A review of the volume indicates that the author has not understated his purposes or, in fact, the attainment of his objectives.

Of the sixteen chapters five are by Dr. Shattuck alone, and the others as specified herewith:

Disorders of the Circulatory System.
Nephritis.
Acute Infectious Diseases.
Acute Infections Most Common in Childhood (Edwin H. Place).
Acute Infections of the Respiratory Tract (G. C. Shattuck and Gerald Blake).
Pulmonary Tuberculosis (John B. Hawes, 2d).
Asthma (Francis M. Rackemann).
Gastro-Intestinal Disorders.

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Syphilis (C. Morton Smith).
Lead Poisoning (Joseph C. Aub).
Anemia (George R. Minot).
Diabetes Mellitus (Benjamin H. Ragle).
Endocrine Disorders (Charles H. Lawrence).
Preoperative and Postoperative Medical Treatment (Charles H. Lawrence).
Vaccine Therapy (Charles H. Lawrence).
Medication.

The last chapter presents an able discussion of, first, a list of eleven "very valuable drugs" (26 pages) and, second, a list of fifteen "useful drugs" or drug groups. Both the apothecaries' and the metric systems are given.

An analytic style is followed in each chapter. For example, in the chapter on "Disorders of the Circulatory System," the order is: pathology and diagnosis; types of cardiac disease—congenital, infectious, syphilitic, degenerative, and myocardial weakness; general principles of treatment for cardiac insufficiency—rest, depletion, medication, diet, and convalescence; supplementary principles and methods (for each of the principal afflictions of the heart); the whole ending with "surgical shock."

The volume contains some *prophylaxis* or *prevention*, although only the cardinal items are listed and the discussion is quite brief.

In the matter of *treatment*, both general and special, the volume appears comprehensive, conservative, and quite up to date, as can be seen from the handling of such subjects as vaccine therapy, hyposulphite of soda to combat metal poisoning, the latest accepted methods for administering arsphenamine, scarlet fever vaccines, and serum therapy, etc.

Much is presented in terse statements or outlines such as the following on page 52: "The technique of isolation (scarlet fever) is that of surgical asepsis reversed. It aims to limit infection to a small zone instead of to keep a small area free from infection;" and on page 37, where a complete working synopsis for the treatment of infectious diseases in general is combined under nine headings, with subdivisions, on a single half page, each heading then being taken up in detail for the major infectious diseases.

We note omission of some items such as prophylactic injections for tetanus, "concurrent" disinfection, the milder chlorine disinfectants, and sufficient cross referencing between the chapters by different authors; moreover, the index is rather brief. There is a minimum of repetition, however, and typographical errors are very rare.

To industrial physicians, surgeons, and hygienists, the brief chapter ($2\frac{1}{2}$ pages) on "Lead Poisoning" by Dr. Joseph C. Aub is very welcome in a book devoted to medical treatment and is surprisingly complete, including a diet list, but we wish that the remaining blank $1\frac{1}{2}$ pages at the end of the chapter might have been devoted to a discussion of the pathogenesis of lead poisoning as Dr. Aub and his group worked it out at the Harvard School of Public Health. These same professions will likewise appreciate, among others, the chapter on "Anemia" by Dr. George R. Minot in which the subject is treated under the headings: types, rest, hygienic environment, diet and care of the digestive system, massage, medica-

ments, splenectomy, and blood transfusions. To the last heading a considerable discussion is devoted.

The book is well balanced and one gets the impression on every page that much is said in little space and by authoritative and experienced persons.—*Emery R. Hayhurst.*

PROCEDURES IN EMPLOYMENT PSYCHOLOGY. A MANUAL FOR DEVELOPING SCIENTIFIC METHODS OF VOCATIONAL SELECTION. By *Walter Van Dyke Bingham*, Director, Personnel Research Federation; Editor, Journal of Personnel Research; Formerly Professor of Psychology, Carnegie Institute of Technology; Consultant in Industrial Psychology: and *Max Freyd*, Research Associate, Personnel Research Federation; Formerly Instructor in Psychology, University of Pennsylvania. Flexible leather. Pp. xi, 269 including references, tables, and index. Chicago and New York: A. W. Shaw Co.; London: A. W. Shaw & Co., Ltd., 1926.

In many respects this volume is a handbook of methods and presents a variety of procedures that have been tested by time and been found to have practical value. The authors have had considerable practical experience in industry and business, and their judgments may be accepted with confidence.

The field as a whole is treated as adequately as the limitations of a single volume permit. There are nineteen chapters dealing with such topics as job analysis, criteria of vocational success, choice of workers to be studied, analysis of the worker, selection of examinations, psychologic tests, rating scales, questionnaires, and test administration and evaluation.

The authors are moderate in their expression of opinion of the value of psychologic procedures, particularly of psychologic tests. They fail, however, to give much detail to the method of evaluation of test scores and to the relation that test scores bear to other factors that surely operate in a total situation.

Two or three chapters are largely given over to a statistical methodology involved in employment psychology. While the number of statistical procedures and formulae that have been included are far from being a complete list, the reviewer is of the opinion that much more than is necessary or desirable has been offered. The presentation of formulae without proofs, without demonstration of their derivation, and without making clear the assumptions upon which they are based, is a dangerous practice. The utilization, also, of complex formulae in such a difficult field as psychologic measurement is generally precarious. Most of those who have attained success in psychologic work in business and in industry have done so with a modicum of statistical procedure.—*Henry B. Elkind.*